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# JOURNAL
## *of the*
# American Veterinary Medical Association

FORMERLY

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(Original Official Organ U. S. Vet. Med. Ass'n.)

EDITED AND PUBLISHED FOR

The American Veterinary Medical Association

INDEX NUMBER

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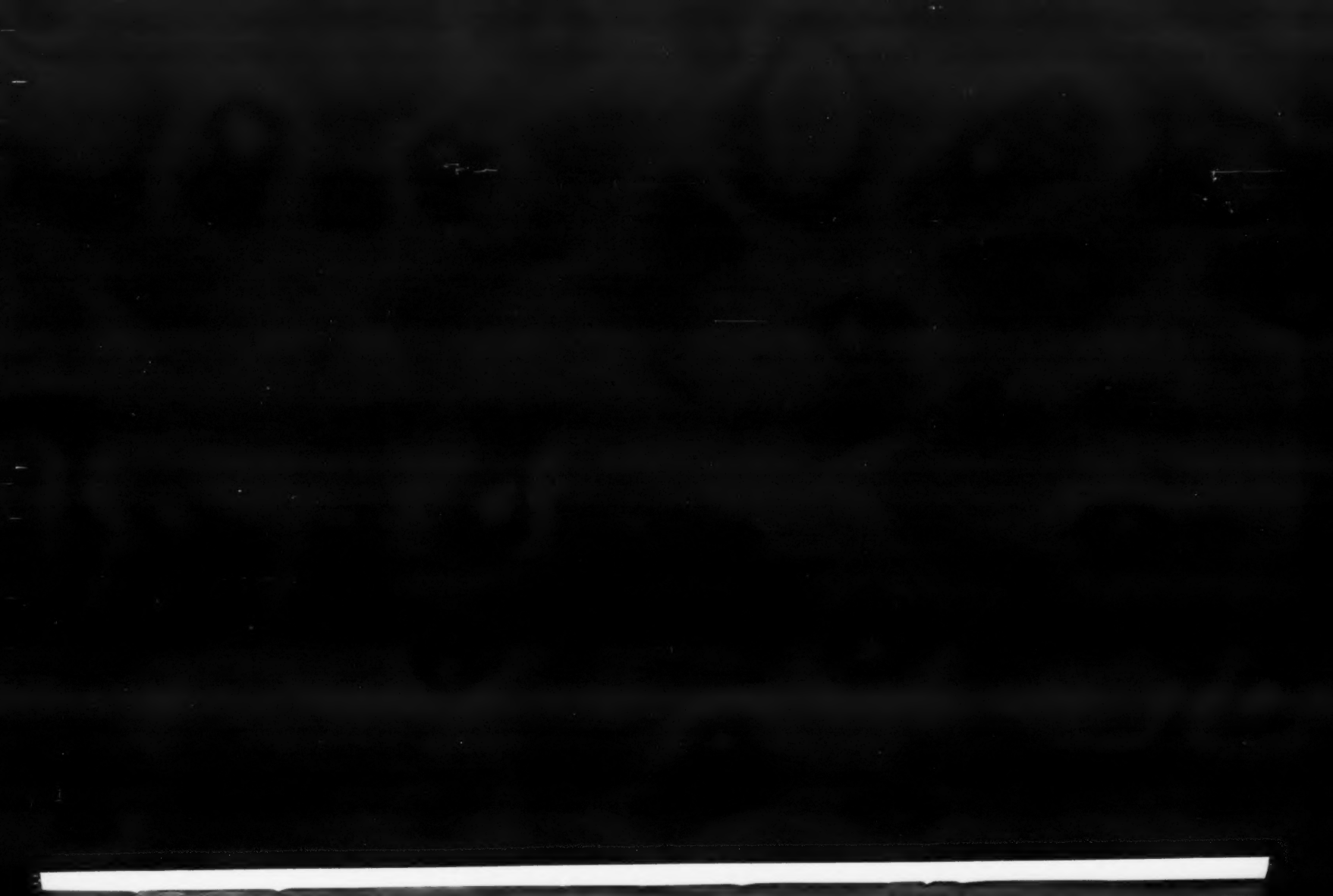

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Vol. LXXVIII, N. S. Vol. 31 June, 1931 No. 6


## IMPORTANT QUESTIONS CONFRONT EDUCATORS

Before another issue of the JOURNAL makes its appearance, approximately 200 additional names will have been enrolled on the roster of graduate veterinarians in this country, bringing the number of graduates of veterinary colleges, in the United States and Canada, past and present, above the 19,600 mark. The increment to the veterinary population this year is the largest since 1923, but even so, the number falls far short of the number estimated by the A.V.M.A. Committee on Education as necessary to maintain the numerical strength of the veterinary profession at the 1930 level.

There is now, and there probably always will be, some difference of opinion among members of the profession as to the number of veterinarians needed to take care of the requirements in this country. The opinion of each veterinarian is quite likely to be influenced by just what his own experience has been. Regardless of opinions on the quantity of veterinarians needed, there is no difference of opinion when it comes to quality. Practically everyone who has given the matter any serious thought is of the opinion that what we need as much as anything else is a better trained veterinarian. Briefly stated, it means that quality is what should concern us, rather than quantity. There are those who

even go as far as to say that if we insure quality, quantity will take care of itself. One thing is certain, and that is that we do not want to increase the quantity of veterinarians if this must be done at the expense of quality.

Our veterinary colleges constitute the key to the situation at the present time. Much light on our veterinary educational system has been shed by the investigations conducted during recent years by our own Committee on Education and other agencies. It is now in order for our colleges to interpret the findings and translate them into action. Two questions are uppermost at the present time. Should the curriculum be lengthened? Should the entrance requirements be raised? A movement is on foot to have a conference of the veterinary deans at Kansas City, and it is to be hoped that these men will be able to arrive at answers to both of these questions.

## MAJOR DUNKIN VISITS AMERICA

Major G. W. Dunkin, M.R.C.V.S., D.V.H., of the Farm Laboratories, National Institute for Medical Research, Mill Hill, England, arrived in the United States on May 5. He will spend about two months on this side of the water and his itinerary includes New York, Boston, Washington, Indianapolis, Chicago, Detroit and Toronto.

The primary object of the visit of Major Dunkin is to study methods used for the production of veterinary biological products in this country, with particular reference to canine distemper. We are advised that Major Dunkin brings with him messages from the President, Council and members of the National Veterinary Medical Association of Great Britain and Ireland, to the American Veterinary Medical Association, conveying fraternal greetings.

When Major Dunkin landed in New York City, he was met by a committee representing the Veterinary Medical Association of New York City, consisting of Dr. Adolph Eichhorn, Dr. Robert S. MacKellar and Dr. E. R. Blamey. Major Dunkin was the guest of honor at a special meeting of the Veterinary Medical Association of New York City, held at the Academy of Medicine, Thursday evening, May 14. He planned to be in Springfield, Ill., on May 27, for the meeting of the Illinois State Veterinary Medical Association. The members of the Southeastern Michigan Veterinary Medical Association have made plans to entertain Major Dunkin while he is in Detroit.

From Detroit, Major Dunkin will go to Toronto and possibly other Canadian cities, including Ottawa, Quebec and Montreal. He plans to arrive back in England on or about June 27. We hope to publish reports of the several meetings attended by Major Dunkin while in this country.

## CONVENTION PLANS WELL IN HAND

Everything in connection with the plans for the Kansas City convention is moving smoothly. The Local Committee on Arrangements seems to have all matters pretty well in hand, according to advices received from the chairman, Dr. A. T. Kinsley.

Prospective exhibitors, who have not yet made reservations for exhibit space at the convention, should do so without delay. Communications on the subject should be addressed to Dr. Ashe Lockhart, 800 Woodswether Road, Kansas City, Mo. It is suggested also that those who have been in the habit of making the arrangements for the alumni dinners should get in touch with Dr. J. D. Thrower, 211 Central Ave., Kansas City, Kan., and give him a preliminary estimate of the number of alumni who will attend each meeting, so reservations may be made for suitable meeting places.

A tentative outline of the program for the week of the convention is shown herewith. This will given our members some idea of how the program will shape up, and perhaps enable some to complete their plans for the meeting. Members of both standing and special committees are urged to arrive in Kansas City not later than Monday morning, if possible, so that each committee

*Tentative Program*

| | Monday Aug. 24 | Tuesday Aug. 25 | Wednesday Aug. 26 | Thursday Aug. 27 | Friday Aug. 28 |
|---|---|---|---|---|---|
| Morning | Meetings of Committees | Opening Session | Sectional Meetings | Sectional Meetings | Clinic |
| Afternoon | Meeting of Executive Board | General Session (Business) | General Session (Business) | General Session (Business) | Clinic |
| Evening | State Association Conference | Alumni meetings, President's Reception and Dance | Banquet | General Session (Papers) | |

may hold a meeting sometime during the day. We always have difficulty in getting these committees together after the convention is under way, and with that thought in mind, Monday morning, August 24, is being specifically set aside for committee meetings, preceding the meeting of the Executive Board, in the afternoon.

## EXECUTIVE BOARD ELECTIONS

A great deal of interest is being shown in the Executive Board elections being held in Districts 6 and 8. Active campaigns are being waged in behalf of a number of the candidates by their admirers and supporters. In District 6 the large California vote is split among three candidates. Likewise, in District 8, Missouri has three names on the ticket, resulting in a division of the support for the Missouri candidates. This is likely to be more of a factor in the final result in District 8 than in District 6, as Missouri does not have the advantage of the large membership that California enjoys. Two of the candidates in District 8 have been placed in the field for president of the A. V. M. A., by their respective states. As a result, the political pot is boiling again.

Quite a few ballots are still out in both districts. These should be voted immediately. The polls will be closed on June 28.

### International Veterinary Congress

President Hall has announced the following additional appointments to the Special Committee on International Veterinary Congress:

D. M. Campbell, 75 E. Wacker Drive, Chicago, Ill.
H. Preston Hoskins, 537 Book Bldg., Detroit, Mich.
J. V. Lacroix, 1817 Church St., Evanston, Ill.
T. E. Munce, Department of Agriculture, Harrisburg, Pa.
B. T. Simms, Oregon State Agricultural College, Corvallis, Ore.

The vacancy on the Permanent Commission of the Congress, created by the death of Dr. H. E. Bemis, has not been filled.

### Doctor Mohler Again Honored

On a recent visit to Havana, Cuba, Dr. John R. Mohler, chief of the U. S. Bureau of Animal Industry, was the guest of honor at an extraordinary session of the National Veterinary Association of Cuba and was elected to honorary membership in the organization on that occasion.

## APPLICATIONS FOR MEMBERSHIP

(See January, 1931, JOURNAL)

FIRST LISTING

BARSTOW, IVAN L. Box 309, Moscow, Idaho
D. V. S., Kansas City Veterinary College, 1910
Vouchers: G. W. McNutt and E. M. Gildow.

BOONE, RALPH WESLEY De Soto, Kans.
D. V. M., Kansas State Agricultural College, 1924
Vouchers: Edwin J. Frick and Edward R. Frank.

CATLETT, JAMES GARLAND 2418 N. Miami Ave., Miami, Fla.
D. V. M., U. S. College of Veterinary Surgeons, 1916
Vouchers: A. T. Knowles and J. H. Yarborough.

CRAWFORD, ANDY Heidelberg Hotel, Jackson Miss.
D. V. M., Kansas State College, 1930
Vouchers: W. O. Hughes and R. H. Stewart.

ELSON, R. E. Vinton, Iowa
D. V. M., Iowa State College, 1917
Vouchers: J. W. Griffith and C. H. Stange.

GUINN, CLOYDE L. Dome of Federal Bldg., Saint Louis, Mo.
D. V. M., Kansas State Agricultural College, 1930
Vouchers: T. B. Pote and E. A. Garleb.

HOFFMASTER, WILLIAM DEAN 321 S. 4th Ave., South Saint Paul, Minn.
D. V. M., Colorado Agricultural College, 1930
Vouchers: G. E. Totten and M. E. Schwab.

HOOVER, EARL FREMONT 4328 Walnut St., Kansas City, Mo.
D. V. M., Kansas State Agricultural College, 1924
Vouchers: Edwin J. Frick, Lawrence O. Mott and J. C. Flynn.

HURTIG, VICTOR CARL Courtland, Kans.
D. V. M., Kansas State Agricultural College, 1926
Vouchers: E. J. Frick and R. R. Dykstra.

KOLL, HARRY 2017 Texas St., El Paso, Texas
M. D. C., Chicago Veterinary College, 1907
Vouchers: N. F. Williams and H. L. Darby.

KHUEN, EDWARD CHARLES 2366 Milwaukee Ave., Chicago, Ill.
D. V. M., Ohio State University, 1920
Vouchers: Leo T. Kilfoy and J. V. Lacroix.

LUCKEROTH, JOSEPH CLEMENCE Seneca, Kans.
D. V. M., St. Joseph Veterinary College, 1921
Vouchers: R. F. Coffey and Edwin J. Frick.

QUIST, DAVID GUNNARD Ogden, Iowa
D. V. M., Iowa State College, 1930
Vouchers: Louis D. Mersch and J. M. Wineinger.

RIESTER, FRANK H. Buechel, Ky.
V. S., Indiana Veterinary College, 1901
Vouchers: E. Calldemeier and J. B. Way.

SMITH, GEO. I. Box 454, Sedan, Kans.
D. V. S., Western Veterinary College, 1899
Vouchers: Edwin J. Frick and R. R. Dykstra.

THOMSON, WILLIAM MAXWELL N. Y. State Vet. Coll., Ithaca, N. Y.
D. V. M., Cornell University, 1916
M. S., Cornell University, 1930
Vouchers: Herbert L. Gilman and C. E. Hayden.

TICE, HARRY R. Summerfield, Kans.
D. V. S., Kansas City Veterinary College, 1906
Vouchers: Edwin J. Frick and R. R. Dykstra.

## Applications Pending

SECOND LISTING

Barber, Charles Brown, 125 S. 4th Ave., South Saint Paul, Minn.
Browne, William Arthur, Cottonwood Falls, Kans.
Brownlee, Hal, Sabetha, Kans.
Butler, Clair L., c/o A. S. P. C. A., 24th St. and Ave. A, New York, N. Y.
Cox, Andrew B., Rose Hill, Kans.
Farr, Jesse Russell, 838 Chafee Ave., Augusta, Ga.
Hodgson, Harold Bishop, 130 Hall St., Athens, Ga.
Horcher, Chas. H., 1414 Queen City Ave., Tuscaloosa, Ala.
Jungherr, Erwin, Storrs Agr. Exp. Sta., Storrs, Conn.
Lyon, Heath L., Hillsville, Va.
Mock, Isaac Tennyson, Little River, Kans.
Nelson, Spencer Kingsley, Box 607, Albert Lea, Minn.
Pfister, B. F., 4322 Main St., Kansas City, Mo.
Phalares, George Spyron, Box 61, Chesaning, Mich.
Picht, Clyde W., 1307 E. 13th, Salt Lake City, Utah.
Spain, Robert J., 1299 Grand Ave., Saint Paul, Minn.
Stanley, Nicholas David, Hope, Kans.
Theiss, Eugene Ware, 117 E. Lexington St., Independence, Mo.
Thompson, John B., 1137 Rowland Ave., Kansas City, Kans.

The amount which should accompany an application filed this month is $7.91, which covers membership fee and dues to January 1, 1932, including subscription to the JOURNAL.

## COMING VETERINARY MEETINGS

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. June 1, 1931. Dr. H. Preston Hoskins, Secretary, 537 Book Bldg., Detroit, Mich.

Texas, State Veterinary Medical Association of, and A. & M. College of Texas Short Course for Veterinarians. A. & M. College of Texas, College Station, Texas. June 1-5, 1931. Dr. D. Pearce, Secretary, Box 335, Leonard, Texas.

Southeast Georgia Veterinary Association. Quitman, Ga. June 3, 1931. Dr. Hugh F. Arundel, Secretary, Box 68, Quitman, Ga.

New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. and 103rd St., New York, N. Y. June 3, 1931. Dr. John E. Crawford, Secretary, 708 Beach 19th St., Far Rockaway, Long Island, N. Y.

American Association of Medical Milk Commissions, Inc. Adelphia Hotel, Philadelphia, Pa. June 8-9, 1931. Dr. Harris Moak, Secretary, 360 Park Place, Brooklyn, N. Y.

Chicago Veterinary Medical Association. Atlantic Hotel, Chicago, Ill. June 9, 1931. Dr. C. L. Miller, Secretary, 508 S. Humphrey Ave., Oak Park, Ill.

Kansas City Association of Veterinarians. Baltimore Hotel, Kansas City, Mo. June 9, 1931. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Tulsa County Veterinary Association. Tulsa, Okla. June 11, 1931. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Oklahoma Veterinary Medical Association. Oklahoma A. & M. College, Stillwater, Okla. June 15-17, 1931. Dr. C. H. Fauks, Secretary, 1919 W. Ash St., Oklahoma City, Okla.

California State Veterinary Medical Association. Pasadena, Calif. June 16-18, 1931. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. June 17, 1931. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

Vermont Veterinary Medical Association. Hotel Vermont, Burlington, Vt. June 19-20, 1931. Dr. G. N. Welch, Secretary, 43 Union St., Northfield, Vt.

Michigan State Veterinary Medical Association. Lansing, Mich. June 23-24, 1931. Dr. E. K. Sales, Secretary, 535 Forest St., East Lansing, Mich.

North Carolina State Veterinary Medical Association. Winston-Salem, N. C. June 24-25, 1931. Dr. J. Howard Brown, Secretary, Rich Square, N. C.

Maryland State Veterinary Medical Association. College Park, Md. June 24-25, 1931. Dr. E. M. Pickens, Secretary, College Park, Md.

New York State Veterinary Medical Society. Syracuse, N. Y. June 25-26, 1931. Dr. C. E. Hayden, Secretary, 110 Irving Place, Ithaca, N. Y.

Central New York Veterinary Medical Association. Syracuse, N. Y. June 25-26, 1931. Dr. W. B. Switzer, Secretary, R. 5, Oswego, N. Y.

Wisconsin Veterinary Medical Association. Sturgeon Bay, Wis. June 29-30, 1931. Dr. B. A. Beach, Secretary, Agricultural Hall, University of Wisconsin, Madison, Wis.

Minnesota State Veterinary Medical Society. University Farm, Saint Paul, Minn. July 2-3, 1931. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

North Dakota Veterinary Medical Association. Fargo, N. Dak. July 6-7, 1931. Dr. Lee M. Roderick, Secretary, N. Dak. Agr. Coll., State College Station, Fargo, N. Dak.

Maine Veterinary Medical Association. Bangor, Me. July 8, 1931. Dr. L. E. Maddocks, Secretary, R. F. D. 2, Augusta, Me.

Kentucky Veterinary Medical Association. Seelback Hotel, Louisville, Ky. July 8-9, 1931. Dr. J. R. Stifler, Secretary, Lebanon, Ky.

New Jersey, Veterinary Medical Association of. Monterey Hotel, Asbury Park, N. J. July 9-10, 1931. Dr. John G. Hardenbergh, Secretary, c/o Walker-Gordon Lab. Co., Plainsboro, N. J.

Virginia State Veterinary Medical Association. Richmond, Va. July 9-10, 1931. Dr. I. D. Wilson, Secretary, Virginia Polytechnic Institute, Blacksburg, Va.

South Carolina Association of Veterinarians. Charleston, S. C. July 14-15, 1931. Dr. G. J. Lawhon, Secretary, Hartsville, S. C.

Western New York Veterinary Medical Association. Webster, N. Y. July 16, 1931. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

Montana Veterinary Medical Association. Bozeman, Mont. July 22-23, 1931. Dr. Hadleigh Marsh, Secretary, Agr. Exp. Sta., Bozeman, Mont.

Northwest Veterinary Medical Association. Corvallis, Ore. July 28-30, 1931. Dr. Clifford Ackley, Secretary, Winlock, Wash.

## STATE BOARD EXAMINATIONS

Pennsylvania State Board of Veterinary Medical Examiners. U. of P. Veterinary School, Philadelphia, Pa. June 12-13 1931. Dr. H. W. Barnard, Secretary, Lancaster, Pa.

Oklahoma State Board of Veterinary Examiners. A. & M. College, Stillwater, Okla. June 15, 1931. Dr. Walter H. Martin, Secretary, 101 S. Evans, El Reno, Okla.

Michigan State Board of Veterinary Medical Examiners. State Office Bldg., Lansing, Mich. June 15-16, 1931. Dr. C. H. Clark, Secretary, 720 State Office Bldg., Lansing, Mich.

Iowa Veterinary Medical Examining Board. State House, Des Moines, Iowa. June 17-18, 1931. Dr. Peter Malcolm, Division of Animal Industry, State House, Des Moines, Iowa.

Massachusetts Board of Registration in Veterinary Medicine. State House, Boston, Mass. June 30-July 1, 1931. Dr. E. W. Babson, Secretary, Gloucester, Mass.

# VETERINARY PARASITOLOGY IN THE UNITED STATES AND IN RUSSIA

*By* MAURICE C. HALL, *President*

*American Veterinary Medical Association*
*Washington, D. C.*

With the rapidly accelerating growth of interest in veterinary parasitology in the United States, it is in order to compare the development of that subject in this country with that in other countries. In general, most of the academic colleges and universities in which attention is paid to parasitology do not concern themselves with veterinary parasitology as such, but confine their attention to morphology, taxonomy and life histories of parasites of animals other than domesticated animals, as a rule. Consequently a comparison of developments in veterinary parasitology must consider primarily developments in state medicine involving the work of governmental units or government-supported units.

Among the larger countries of the world, the two which are paying the most attention to the subject of veterinary parasitology, as developed in units which are under some form of governmental control, are the United States and Russia. A comparison of the conditions in the two countries mentioned is of interest as showing the organization and lines of attack on problems in veterinary parasitology and the relative importance which attaches to the subject in these countries.

## CONDITIONS IN THE UNITED STATES

*The Federal Bureau of Animal Industry:* The major unit for research in veterinary parasitology in the United States has been for many years the Zoölogical Division of the Bureau of Animal Industry, U. S. Department of Agriculture. That division originated in the appointment of Dr. Cooper Curtice, in charge of parasitological investigations, on August 1, 1886. From that time until his resignation on May 31, 1891, Dr. Curtice accomplished a number of very important things, including a study of the life history of the ox warble, the essentials of the life history of the cattle fever tick, the description of the nodular worm of sheep with the evidence that it causes nodular disease, and the publication of a bulletin on sheep parasites which is a classic in

its field. Dr. Curtice was also a pioneer in urging that the cattle tick be investigated as a possible transmitter of Texas fever, in urging and beginning tick eradication, and in urging and beginning eradication work in bovine tuberculosis. Subsequent to his resignation in 1891, he returned to the Bureau and did excellent work in the control of sheep parasites. It was with great regret that the Zoölogical Division saw this pioneer and veteran of over 40 years' fighting against parasites and diseases of live stock retire on September 1, 1930.

Before Dr. Curtice left in 1891, Dr. Albert Hassall, a pupil of Cobbold, was transferred to the Zoölogical Laboratory on March 7 of that year, and Dr. Charles Wardell Stiles, a pupil of Leuckart, was put in charge of the laboratory on June 3. This same year the index-catalog of veterinary and medical zoölogy, a work of inestimable value, was begun. From that time to 1902, these two men, with very little assistance of any sort, turned out an immense amount of valuable work, culminating in the description by Stiles of the new human hookworm, *Necator americanus*, and his demonstration of the extent and importance of hookworm disease in the South, and followed by his transfer to the Public Health Service as Chief of the Division of Zoölogy of the Hygienic Laboratory.

On June 1, 1903, Dr. Brayton H. Ransom was put in charge of the Zoölogical Division. Individually he did a large amount of very sound and careful work on such diverse topics as bird tapeworms, trichinosis, cysticercosis, meat inspection problems in parasitology, nematodes of ruminants, standardizing the arsenical dip for cattle ticks, the life history of *Ascaris suum*, and the development of the swine sanitation system. At the same time he began the expansion of the Division to take care of the many problems which confronted it. At the time of his death, September 17, 1925, the Division had twelve scientific employees in Washington and at four field stations.

After Dr. Ransom's death, the writer was put in charge of the Zoölogical Division, and the expansion of the Division was continued. At the present time the Division has 36 professional and 5 subprofessional employees in Washington and at nine field stations, engaged in work on nine major projects (administration, index-catalog and collections, poultry parasites, swine parasites, ruminant parasites, horse parasites, miscellaneous parasites, treatment, and extension work.

The Meat Inspection Division of the Bureau takes cognizance of numerous parasites, such as cysticerci, hydatids, trichinae, and kidney worms, in its routine procedures. One way and another it is a very important agency in parasite control.

The Tick Eradication Division is devoted entirely to parasite eradication, and it is sufficient to say that it has wiped out ticks and piroplasmosis over an area of over 600,000 square miles.

The Field Inspection Division deals mostly with parasites, as its major line of work in the field is the control of scabies and dourine, and the presumption is that new lines of parasite control which the Bureau may delegate to some division for large-scale operations will fall to this Division.

*Federal Bureau of Entomology:* In the federal Bureau of Entomology there is a unit devoted to the investigation of external parasites of man and animals. This is a unit of fifteen entomologists and two agents at Washington and at eleven field stations. It has done a large amount of valuable work on insects, ticks and mites affecting domesticated animals, including poultry.

*Federal Bureau of Biological Survey:* The Bureau of Biological Survey has a fur-farm at Saratoga Lake, N. Y., at which it investigates diseases of fur-bearing animals, including a consideration of parasites. Its field forces take cognizance of diseases and parasites in wild animals, and the problems arising in this connection are largely handled by coöperative arrangements with other federal agencies.

*Federal Forest Service and National Parks Service:* The field men of the Forest Service (U. S. Department of Agriculture) and of the National Parks Service (Department of Interior), like the field men of the Biological Survey, take cognizance of diseases and parasites of wild animals, and the problems of control are usually handled through coöperative arrangements with other federal agencies.

*Federal Bureau of Fisheries:* The Bureau of Fisheries (Department of Commerce) has one man working on fish diseases as a full-time assignment, and this work includes some consideration of parasites.

*Agricultural experiment stations:* In a recent publication of the Department of Agriculture, Miscellaneous Publication No. 89, there are listed the problems in parasitology on which the state experiment stations are engaged, and this publication affords evidence of the growing interest in parasitology. The problems

investigated are outlined below by states and insular possessions.

*Porto Rico:* General survey of parasites of domesticated animals; liver flukes of sheep, goats and cattle; swine hookworms; swine kidney worms. *Guam:* Stable flies. *Kentucky:* Animal parasites. *Wisconsin:* Animal parasites. *West Virginia:* Stomach worms and lung worms of sheep and cattle. *Maryland:* Swine parasites; repellents for stable flies. *California:* Poultry parasites; Hippelates flies. *Kansas:* Resistance to parasites; resistance of poultry to parasites; biology of chicken nematodes and cestodes; embryology of chicken cestodes; house flies as carriers of chicken cestodes. *Michigan:* Life history and control of pathogenic parasites of food animals. *North Dakota:* Control of intestinal worms; internal parasites of sheep; roundworms in swine; internal parasites of poultry; lice; mites. *Wyoming:* Life history of *Moniezia expansa;* of *Thysanosoma actinioides;* of *Sarcocystis tenella. Oregon:* Liver flukes and lungworms in sheep and goats; salmon poisoning of dogs. *Texas:* Control of stomach worms in sheep and goats; screw worm and wool maggots; goat lice; sheep scab. *North Carolina:* Treatments for stomach worms of sheep. *Illinois:* Swine sanitation; ox warbles. *Louisiana:* Control of roundworms in swine. *Florida:* Swine kidney worms; Manson's eyeworm; life history of intermediate host of Manson's eyeworm. *Minnesota:* Endoparasites of domesticated animals; source of infection of domesticated foxes by tapeworms; flukes of the genus Collyriclum as poultry parasites; horse flies. *New Jersey: Ascaridia perspicillum;* mosquito control. *Oklahoma:* Poultry cestodes. *New York:* Horse flies. *Arkansas:* Horse flies. *Indiana:* Fly control in dairy barns. *Montana:* Mosquitoes. *Connecticut:* Mosquitoes.

From the foregoing it appears that 25 of our states and insular possessions are carrying on research work in veterinary parasitology, with the parasites of poultry, swine and sheep as the preferred projects. Over half of the states have no such work, but within the next decade there will probably be a veterinary parasitologist in practically every state, as well as in every territory and insular possession.

*Extension Service:* In the Extension Service there are a number of projects, listed below by states, dealing primarily with veterinary parasitology.

*South Dakota:* Swine sanitation. *North Dakota:* Swine sanitation; sheep parasites; poultry sanitation. *Maine:* Blackhead control; poultry sanitation. *Kentucky:* Animal parasites.

*Kansas:* Swine sanitation; poultry parasite control. *Iowa:* Swine sanitation; poultry parasite control; bot control. *Illinois:* Poultry parasite control; swine parasite control; cattle parasite control; sheep parasite control. *Georgia:* Swine sanitation; poultry parasite control; animal parasite control.

Eight states have formal extension projects in the control of parasites of live stock, with the control of parasites of swine and poultry as the most common projects. Probably every state should have such projects and doubtless most of them will have before long.

### Conditions in Russia

The status of veterinary parasitology in Russia has recently been discussed by Dr. K. I. Skrjabin, Director of the Institute of Parasitology of the Commissariat of Agriculture. His paper on this subject appeared in the monthly bulletin, v. 3, no. 6, for May-June, 1930, of the Office International des Epizooties created by the international arrangement of January, 1924, and was reviewed by Zunker in the *Berliner Tieraertzliche Wochenschrift*, v. 46, no. 52, of December 26, 1930. It is a comprehensive summary of the present situation and the plans for the future.

*Organization:* Skrjabin contends that up to the present there have been but scattering and inadequate attacks on the problems of control of worm parasites, and argues for large scale and concerted action. As a preliminary to this in Russia, the Institute of Parasitology was established at Moskow as a central agency for the study of scientific and applied helminthology, and for the direction of the institutes scattered about Russia. Chairs of parasitology, with special emphasis on helminthology, were established in the fourteen higher veterinary schools, thus providing for research and assuring the training of qualified veterinary parasitologists, workers not previously available. As a principle of procedure, complete parasitological necropsies were made the basis of the work, a thing for which the present writer has contended for some years as the only sound method of teaching veterinary parasitology. In the course of eleven years, about 50,000 necropsies have been carried out in this manner, thereby establishing in a fairly sound way the nature of the parasite fauna of the country and giving some indications as to the more important infested areas.

Up to the end of 1929, the government had sent out 75 expeditions covering the country from end to end, and had established

over 50 medical, veterinary and zoölogical institutes for helminthology. The more valuable specimens collected in these institutes or by expeditions are deposited in the central museum at Moskow, and there are now approximately 200 type specimens of new species in that collection. Up to this time about 2,000 physicians have been trained in helminthology. (At the recent first Congress of Helminthologists at Moskow, 168 official delegates were present.) About 300 papers on medical and veterinary helminthology have appeared from the institutes of helminthology, a statement which the American parasitologist, who is confronted with the necessity for finding out what is concealed in the Russian in which most of these papers are published, will not be disposed to deny, as he receives a flood of important papers in that baffling language. It may not be a digression to say at this point that the Washington staff of the Zoölogical Division reads twelve languages and speaks eight, but has only three persons who read Russian and only one person who speaks it.

*General control measures:* Skrjabin divides the helminthiases into the geohelminthiases and the biohelminthiases, the first including those caused by monoxenous worms without intermediate hosts and with infection carried in soil, water, etc., and the second including those caused by heteroxenous worms using intermediate hosts and with infection conveyed through eating intermediate hosts or through the attack of such intermediate hosts as ticks and biting flies. He regards the biohelminthiases as the easier to control, by virtue of the possibility of breaking through the life history of a parasite by an attack on the intermediate hosts, a general principle which is open to argument so far as ease of control is concerned. As agencies of control for helminths he would utilize a large network of helminthological institutes, abattoir meat inspection service, local commissions, and the zoötechnic organizations of stockmen and farmers, to follow a program which would be specific, periodical, rational, obligatory and predetermined. As specific measures he advocates war on vagrant dogs, on various species of mollusc intermediate hosts, rats, and flies, and the drainage of wet pastures.

*International program:* All of this, he contends, should be carried out as an international program, believing that countries in general have ignored the importance of worm parasites which have risen to the status of an international menace. His program is briefly stated as: an international entente; application of a coördinated plan; and generalized intervention. Such a campaign would

lead to economic advantages in the provision of sound and healthy animals, not only by relief from parasites but also from parasite effects predisposing to other diseases. It would also diminish parasitism in man by cutting off the source of his many parasites which come to him from lower animals. These economic and medical benefits, he concludes, would thus help in the solution of the great social problem—the production and development of a sound race of mankind.

## Discussion

It is impossible to compare the status of the United States and Russia as regards progress in veterinary parasitology very accurately, since the writer lacks first-hand knowledge of the Russian situation, and there are too many places where there is a lack of published comparative data. However, from what is available, the following impressions are submitted:

The growth of the subject of parasitology in the United States has been slow and steady. In the generation which preceded such veterans as Curtice, Stiles, Hassall, Ward, Linton, Verrill and Pratt, there was only one parasitologist of note in this country, Joseph Leidy. Of his successors named above, only the first three were primarily interested in veterinary parasitology. A few veterinarians, such as Dalrymple, Francis, and some others, did occasional work on parasites of live stock. About fifteen years ago, the tide turned and there was a marked increase in interest in veterinary parasitology, which interest is still increasing and promises to increase for at least another decade. We have come to realize that modern conditions are confronting us with a new problem, and that parasites are becoming more, and not less, troublesome.

The growth of interest in parasitology in Russia has been rapid and spectacular. Previous to the war there were in Russia a few helminthologists, such as Sinitsin. Since the war the number of workers has jumped rapidly, and these workers are able men for the most part, as far as one can judge from their publications. In a general way it appears that in a decade they have developed an organization in this field which is equivalent to what we have developed in 45 years.

Comparing the status of the two countries at present, it appears that the work in the United States is less definitely organized and correlated, that we have fewer trained workers and organized institutions in veterinary parasitology, and that we lack the

statistical data which the Russians have obtained by coördination and field expeditions. On the other hand, we have translated into action a number of programs, such as tick eradication, scabies eradication, dourine eradication, and swine sanitation; if Russia has similar control campaigns actually under way the evidence has not come to the writer's attention, although it is clear that plans are under way for nation-wide control of parasites, and, as noted above, that international plans are being formulated.

From the comparison one may say that Russia probably has a keener appreciation of the problems of parasitology than has the United States, probably because of the influence of Dr. Skrjabin, but it is probable that the growth of interest in this country will soon bring it up to a level with the Russian appreciation. The Russians are in a position to organize and push campaigns for parasite control through the strong central government to an extent not possible in this country, where the states have extensive powers of government. To equal the Russian programs the United States must not only continue the development of its federal agencies for parasite control, but must also develop in the individual states adequate agencies for control, a thing which may confidently be expected as a development of the near future. But it must do more than this. It must coördinate the federal and state agencies in such a way that unnecessary and expensive duplication of work may be avoided as far as possible, and that coöperation may exist wherever possible. Such coöperation must rest primarily not on the basis of formal written agreements, but on the continuation of the spirit of friendliness which has been characteristic of American parasitologists and on an intelligent appreciation of the need for and the value of coöperation.

As specific plans for coöperation the following are worth consideration:

For some years the Zoölogical Division has offered to function as a central taxonomic unit for workers outside of Washington who were not primarily interested in taxonomy. Most of the students of taxonomy outside of Washington are faculty members or graduate students of universities. For efficient taxonomic work one needs adequate library and catalog facilities, large collections, and a force of trained taxonomists with time for identifications, and these are available nowhere in the United States to the extent that they are available in Washington. It is out of the question to duplicate elsewhere the Library of Congress, The Surgeon-General's Library, the Library of the U. S. Department

of Agriculture, and the other libraries at Washington. It is out of the question to duplicate and keep up to date the index-catalog to which Dr. Hassall has devoted forty years, and which now has a staff of four workers—and needs more. It is out of the question to duplicate the collections which by law are units of the U. S. National Museum collection. With these facilities the specialists of the Zoölogical Division can identify, in fifteen minutes, specimens to which a worker outside of Washington and without those facilities might devote months without success. If a man has in mind work other than taxonomic, as is commonly the case at agricultural colleges, experiment stations, and veterinary colleges, it will usually save his time to have the parasites he is interested in identified in the Zoölogical Division in order that he may proceed more promptly to go on with the work in which he is interested. Such work is frequently along the lines of studies in the pathology, treatment or prophylaxis of parasitism, work which cannot be done to advantage in Washington.

Another thing which might be done as a means of obtaining the data which Russia has obtained from its institutes and expeditions would be to have parasite surveys made by workers in the various states in order that we might know what parasites we have in this country and where they are. This work should be supplemented by the inauguration and development of a statistical service in the federal meat inspection work, with a tracing system to follow diseases and parasites to their source.

It is evident that the emphasis which the Bureau of Animal Industry has placed on parasites has not been misplaced, and that in Russia an even greater emphasis has been placed on it. Taking the situation by and large, conditions in this country do not compare unfavorably with those in Russia as regards attention to veterinary parasitology, but the increasing interest in this subject in this country comes at a time when it is needed if we are to handle the subject in a way which will enable us to maintain a level comparable with that of Russia.

As regards Skrjabin's proposal for international action in the control of worm parasites, there are two evident objections. In the first place, there is a certain amount of development of internationalism as evidenced by such things as the League of Nations, the World Court, and Briand's proposal for a United States of Europe, but there is an even more intense growth of nationalism as evidenced by the fascist and communist youth movements and other activities. In the second place, our knowledge of con-

trol measures for worm parasites is still too inadequate to permit of satisfactory control even with the limits of one nation. The palliative measures to which Skrjabin takes exception are all we have. To prevent the international movement of parasites we should need practically 100 per cent perfect control measures, either by treatment or prophylaxis, and we haven't these things. All that can be urged at present is increased research of a basic sort and the provision of efficient channels for translating the results of research into practice as rapidly as possible. International action appears to be a thing of the rather remote future.

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### New Dignity for the Veterinarian

Ohio State University's College of Veterinary Medicine has graduated more veterinarians than any other state institution in the country, according to a survey made by the United States Department of Education. It is the second oldest of the state veterinary colleges.

Veterinary medicine has assumed a new importance during the past few years and its practitioners a new dignity. The old "hoss doctor," with his questionable knowledge of anatomy and therapeutics, has passed out, and in his place there stand scientifically trained men, ready to render invaluable service to one of the nation's fundamental industries.

Before the rise of scientific veterinary practice, the breeding and care of farm animals was more or less haphazard. The monetary loss from disease was enormous. Scourges swept the farm districts unchecked, their source and their remedy deep mysteries to most agriculturists. The quack veterinarian flourished, with his firing-iron, purgation and blood-letting, which represented about the limit of his knowledge.

Now, however, veterinary medicine has been placed upon the same high plane as human medicine. It has its research workers and its specialists, its surgeons and its pathologists. Entrance requirements at the better-class schools have been elevated until they compare favorably with the other learned professions. Courses now are of regulation academic length and a decided improvement in the scientific and social standard of the profession, which dates back to Aristotle, Pliny, Celsus and Galenus is evident.

Editorial in *Columbus (Ohio) Evening Dispatch*

# STERILITY IN COWS CAUSED BY ENDOMETRITIS*

*By* J. F. RANKIN, *Astoria, Ore.*

Sterility of cows, in which endometritis plays a large part, is a serious source of economic loss. In permanent sterility the animal is worthless except for food purposes. Temporary sterility cuts milk production one-half or more and the increase in young is reduced in direct proportion to the length of the sterility period. A cow that has to be maintained on the dairy farm, for twelve to eighteen months without calving, is an unprofitable animal.

A complete history of the animal to be treated should be obtained from the owner. An accurate record of this history, the findings and results of ensuing treatments should be kept. The physical condition of the animal to be treated should be taken into consideration, especially as to condition of flesh and whether or not there is evidence of debilitating disease.

It is very necessary in sterility work to be able to determine whether or not the animal is pregnant. The breeding history given by the owner should be considered, but not entirely relied upon. Experience makes a better diagnostician and for that reason every opportunity should be taken to examine pregnant animals that have been pregnant for a known period. Many times a veterinarian will be called upon to examine animals for pregnancy, as in the case of sales, where animals have been pasture-bred, where records have been lost or where farms change hands.

Before making an internal examination, the external appearances should be noted, observing whether the tail and tuft of the vulva are soiled by vaginal discharges. The operator must have thorough knowledge of the physiology and anatomy of the reproductive organs of the cow. He does not need an elaborate display of expensive instruments. A good vaginal speculum, two pairs of forceps, a uterine dilator, a pair of blunt-pointed shears, two catheters, a pair of dressing forceps and a syringe with a flexible probe-pointed cannula are all that is required in the usual sterility case.

The endometrium or uterine mucosa is very delicate and subject to infection and injuries. The relation of endometritis to

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cervicitis is very close and usually they exist together. The subject of cervicitis will be covered later by Dr. Kingman, who was assigned that subject. Williams states that the histology of endometritis has not been studied and that the bacteriology has been given practically no attention.

In herds badly affected with Bang's disease more cases of endometritis are found. Every aborting cow has endometritis. One author states that many cows that apparently calve normally have a chronic endometritis and that many cases of endometritis are continuations of the endometritis of pregnancy. Other causes are retention of the placental membranes, or particles of them, within the uterine cavity, or injury to the parts by rough manipulation of the uterine walls while removing fetuses or placental membranes, or abrasions or tears in the uterine mucosa by difficult parturition. The infection may be caused at times from the male by copulation. Contributing factors are high protein feeding and lowered resistance. Endometritis is found in only a small percentage of virgin heifers.

### Clinical Manifestations of Endometritis

Clinical manifestations are pus discharging from the vulva, soiling of the tail and hair tufts, and presence of pus in the vagina. On palpation through the rectum the cervical canal is found to be unusually large and rigid. The uterus and horns are large and flabby and lack tone. Usually catarrhal or mucopurulent flakes come through the uterine catheter, induced by irrigation or massage. In less severe or subdued cases the uterus and horns are less flabby and tend to rigidity and there is little or no discharge present. The ovaries should be examined for lesions such as cysts, retained corpus luteum and fibrous conditions.

The object of sterility treatment is to stimulate the uterine mucosa and cause it to function normally. There is nothing especially new or startling to offer about endometritis as a cause of sterility. Physicians use glycerin extensively for the treatment of sterility in women and report good results. I use glycerin incorporated with other drugs commonly used in sterility. Using 3 per cent Lugol's solution in glycerin, which is injected with a syringe having a long, flexible, probe-pointed cannula, through the cervical canal, 40 to 60 and even 100 cc of glycerin and Lugol's solution is injected. In conditions where there is a

large amount of pus, the uterus should first be douched out with a physiologic salt solution before using the glycerin solution.

In the less severe case I use acriflavine incorporated with glycerin (5 to 10 grams of neutral acriflavine powder, dissolved in eight to sixteen ounces of distilled water, and glycerin sufficient to make one gallon). This makes an attractive yellow solution, which is also used for cervicitis.

The uterus is pulled back with the uterine forceps attached to the cervix, and the horns and uterus proper gently massaged with a backward motion. Every precaution should be used to prevent rough manipulation. The ovaries are examined for abnormalities and if cysts are present they are ruptured by pressure and in certain conditions the ovaries are gently massaged. Too much stress on the massaging and treatment of the ovaries, in the belief that this will relieve the cause of sterility, is in my opinion contraindicated in a large percentage of cases. Cysts and diseases of the ovaries are secondary lesions in my opinion. Relieving the inflammatory condition of the uterine mucosa corrects, to a large measure, the ovarian lesions.

A large percentage of severe endometritis cases might be prevented, especially in large herds, under the supervision of a veterinarian, if, after parturition, as shown by the character of the discharge from the vagina (scarlet-gray exudate or grayish yellow pus or tissue débris), douching be employed at regular intervals with a physiologic salt solution. The salt solution should not be strong enough to "pickle the cow."

## Case Reports

Let me give some case reports: A group of 12 cows was examined and one was recommended to be slaughtered. All of this group excepting one had a severe cervicitis and endometritis with a purulent discharge. They were treated weekly for six treatments. Two of these cows disappeared and seven of the remaining eight became pregnant.

In a second group, 21 cows and heifers were examined on May 29, 1929, and are a very interesting group because many of them are four or five years old and have never been known to be pregnant. Three of this group were found to be pregnant, and three were recommended to be slaughtered. Of these three one was a nyphomaniac with severe cervicitis and cystic degeneration. A Graham student had treated her several times previously. The other two had dead fetuses, accompanied by

pyometra, both fetuses lying deeply in the abdominal cavity. The remaining fifteen cows were treated at weekly intervals eight times, and at 3-week intervals four or five times. These animals were very valuable, the mature cows all having butterfat records and the heifers had unusually good breeding. As time prevents giving a detailed report on each of the 21 animals, I have classified them into sub-groups.

The first sub-group consisted of four young cows that had never come in season, according to the history obtained. It is possible they had been in season because they had not been running with the bull and hired help was depended upon to see they were bred. One four-year-old cow had aborted twice, the last abortion being in August, 1928. One five-year-old cow produced but one live calf, in April, 1927. Both these cows had cervicitis and endometritis. The other two heifers, four and five years old, had no history of previous pregnancies. They both had cervicitis and mild endometritis. All four were vaccinated with live-culture abortion vaccine at the time of the first treatment and received two doses of corpus luteum. The solutions of glycerin and Lugol's and glycerin and acriflavine were used in connection with massage. The retained corpora lutea were squeezed out of the ovaries. Three of these cows came in season and became pregnant. Estrum could not be produced in the remaining cow.

The second sub-group consisted of 6 young cows, all four and five years old. Four of them had no previous history of pregnancy, and the other two had aborted once each. Five of these were given live-culture abortion vaccine and all received two doses of corpus luteum. Two of the virgin heifers and one of the cows with an abortion history became pregnant. No results were obtained with three of the cows. Four of this sub-group of six had endometritis and were treated the same as the second sub-group, excepting that in this case the ovaries were not massaged.

The remaining sub-group of five old cows, from five to eight years old, were all sterile for periods of from two to five years. Four had cervicitis and endometritis and the fifth cow had mild pyometra. Three of the cows affected with endometritis became pregnant, following use of massage and the solutions mentioned above. No vaccine or corpus luteum was used. This group of 21 animals is interesting because they were sterile for a long period of time and were in a large herd badly affected with Bang's

disease. Also a large percentage of the virgin heifers from this group aborted after becoming pregnant.

In another group of 20 cows, sterile for from three to twelve months, sixteen had aborted one or more fetuses, most of them during their last pregnancy. On examination, eight were found to be pregnant. Eleven of the remaining twelve had endometritis. After three treatments three were recommended to be slaughtered and the remaining nine were treated at three-week intervals. After six treatments seven of the nine were pregnant. Two cows from this group had large fibrous bands obstructing the entrance to the cervical canal. The bands were severed. A group of five cows was treated and three became pregnant, after four treatments.

A group of six was treated and five became pregnant after five treatments. A group of eight was treated and five became pregnant after six treatments. The last three groups were treated at intervals of three weeks to a month.

(N. B. Discussion of this paper was in conjunction with the following paper, by Dr. H. E. Kingman.—Editor.)

## Life Histories Revealed

The life histories of six poultry parasites, nematodes of the superfamily Spiruroidea, have been demonstrated for the first time in this country by Dr. Eloise B. Cram, zoölogist of the Bureau of Animal Industry, U. S. Department of Agriculture, and are described in Technical Bulletin 227-T, just published. The bulletin, which is intended especially for zoölogists and veterinarians, explains that those nematodes were found to exist in seven intermediate hosts which were heretofore unknown as hosts. These discoveries double the number of life histories known throughout the world for similar nematodes which are important parasites of poultry and game birds.

The bird hosts in which infestations were produced in the experiments described by the author include chickens, ducks, pigeons, bobwhite quail, and ruffed grouse. The intermediate hosts include dung beetles, cockroaches, grasshoppers and isopods. The bulletin describes and illustrates each parasite in the various stages of its development and reports observations on its location in the body at different times, and on the periods necessary for its complete development in the intermediate and the final hosts. It also discusses clinical and pathological effects of the infestations.

# ENDOCERVICITIS IN THE COW*

*By* H. E. KINGMAN, *Fort Collins, Colo.*

*Colorado Agricultural College*

Any slight disturbance of function on the part of the cervix may constitute a mechanical or chemical barrier to the passage of the sperm through the cervix to the uterus and subsequent inhibition of fertilization.

There are no cilia in the cervix to aid the sperm on its way. It must swim through a rough, tortuous route, filled with glassy mucus.

In the case of the mare, semen is ejaculated straight into the uterus, but not so in the cow. On the contrary, the cervix constitutes the first barrier to be crossed. Therefore, it is believed that diseases of the cervix occupy a common place among the causes of sterility in the cow.

Diagnosis is the most important phase of the practice of medicine. Without an accurate diagnosis treatment becomes a "hit and miss" procedure.

Effectively guarding the uterus against invasion from the rear, opening a door, and providing safe passage for the sperm, and later, stretching like a rubber band to permit the passage of the enormous fetus, its fluids and membranes, the cervix is truly a phenomenal structure.

The clinical picture of endocervicitis comprises recognizable changes in shape, size, color, exudate and lumen of the cervix.

Harvey B. Mathews, writing in the *Journal of Surgery*, divides endocervicitis into four groups for the sake of diagnosis and treatment. From my experience it appears that a similar classification should be made in the case of the cow, using a slightly different clinical and pathological picture.

*Group I:* In group I should be placed cervices representing a simple catarrhal reaction as a result of superficial infection. The vaginal os is moderately dilated, through which the first ring is visible as a cherry-red tumor, apparently obstructing the canal. It is not angry, neither is it eroded, but it is swollen and changed from the normal in shape and size and color. Its shape reminds one of a cherry. (It might be illustrated by exposing the tip of one's tongue through one's puckered lips.) Its

*Presented at the sixty-seventh annual meeting of the American Veterinary Medical Association, Los Angeles, Calif., August 26-29, 1930.

size depends upon the age of the cow and the number of pregnancies. In the young cow (group I) the cervix should not be more than one inch in diameter and only slightly larger at the posterior portion than the rest of the cervix.

The lumen appears to be closed as a result of the inflammatory edema but the obstruction does not interfere with the escape of fluids from within and it is a question relative to how much it might stand in the way of the upward passage of the sperm.

In group I the exudate is thin and watery. It may or may not be streaked with pus and neither is it copious, but it renders the membranes moist and slimy.

In studying the exudates of endocervicitis, the path that forms one's chain of thought divides, or perhaps one should say, one's line of reasoning is intersected by two other factors. First, one must be able to recognize the healthy mucus of estrum, a secretion which is copious, viscid, cohesive (not adhesive), clear, fills the open cervix, collects in the vagina, and escapes through the vulva to hang in a string from the commissure. Secondly, the exudate may not originate in the cervix but in the uterus, and constitute a symptom not of endocervicitis, but of endometritis of the first degree, which is escaping through a healthy cervix.

However, one may conclude that the presence of an exudate in the vagina is indicative of either endocervicitis or endometritis and therefore it permits one to make a provisional diagnosis of one or the other, depending upon other evidence, such as anatomical changes to complete the differentiation.

## Treatment

Most cases of this type recover without treatment. However, like a "cold" in the human patient, if endocervicitis of the first group is allowed to continue it may develop into a more serious form.

In group I, the infected area is usually accessible to local treatment, and vaginal douches may prove beneficial. One may use hot sodium chlorid, 2 per cent sodium bicarbonate solution, 2 to 4 per cent mercurochrome, acrifiavine (1-500) in water, glycerin, or gelatin, introduced by means of a long-nozzled syringe or collapsible tube.

Suppositories of cocoa butter containing boric acid, tannic acid and bismuth subgallate may be introduced by means of a special cannula.

Group II is recognized by the everted, sometimes eroded, intensely reddened cervix, with a well-dilated vaginal os, through which protrudes the infected folds of the first cervical ring which, in part, because of its liberation from the confining walls of the cervical canal, acquires greater freedom to enlarge through engorgement with blood and inflammatory edema.

The infection is of longer duration than in the first group, and depending upon the relative size of the cow and genital tract, the cervix is from one to two inches in diameter.

Upon palpation, the posterior part of the cervix feels like a roll of hard tissue at the end of the apparently otherwise healthy cervix.

The exudate varies from a clear, thin watery fluid to a thick tenacious, stringy mass, sometimes clear but more frequently red-brown or slate-gray in color (slate-gray mucus may originate also within the uterus). The exudate is free from odor and is not present in large amounts. It may be streaked with yellow pus as if the pus were from some well-circumscribed area, or the outlet of a duct that is discharging its contents into the cervical canal where it is carried along by the cervical mucus without becoming mixed with it.

### Pyometra May Be Present

Straight yellow pus originating from the cervix is not common. When found in the vagina, one suspects pyometra which may or may not be associated with encocervicitis.

In the human patient it is understood that the discharge constitutes a very important symptom but I do not believe that it is of quite so great clinical significance in the cow, although I would not minimize its importance in diagnosis.

The treatment of group II should be more vigorous than in the milder infection. One resorts to cauterization either with the actual cautery or with silver nitrate. Actual cautery gives more satisfactory results and is easy to apply. Anesthesia is not necessary except in irritable subjects. Then epidural use of procaine stops all resistance and insures more effective and pleasant work.

In large and mature animals the cervix may be drawn to the vulva to be treated, but in young cows this becomes difficult and subjects the cervix to considerable injury by forceps. It is therefore better to treat the cervix through a lighted speculum.

Types bordering between I and II respond to tincture of iodin, 20 per cent mercurochrome, or 1 per cent potassium perman-

ganate, applied by means of a swab, introduced through a lighted speculum.

Wherever cauterization is used it should be followed by vaginal douches of salt water or sodium bicarbonate solution for a period of one week or ten days. For the first few days following cauterization the whole picture is intensified by sloughing, increased exudation and some pain. As these symptoms subside, the tissues approach normality and the "palma plicata" returns to the same size as the rest of the cervix.

Group III is encountered most frequently in aged cows and is recognized through the presence of polypoid growths resulting from engorgement and proliferation of the papillae or terminal endings of the longitudinal folds of cervical mucous membrane.

The infection is confined principally to the first ring, which may or may not be severely edematous nor even excessively red. It takes on the appearance of a fringe with a base or cervical attachment that is narrow (about ¼ inch) and well defined, while the free margin is divided into thick, edematous, red lobes or fimbriae. The vaginal os is found to be an ill-defined circle of mucous membrane, 2½ to 3 inches in diameter, that is pushed aside by the proliferating ring. The finger-like growths at the margin fall over the lumen, occluding it, although when the folds are pushed aside it permits the introduction of part of one finger. With the thumb and finger one may clearly follow the comparatively thin wall of the ring at its union with the body of the cervix.

The exudate consists of a slimy mucus that lodges between the folds, covers the surface and "slops over" into the vagina. If erosions are extensive the exudate is stained with blood and is rust color.

### Ablation Indicated

The treatment must be ablation of the entire ring at its base. This is done by grasping the ring with forceps and separating it from its attachment by means of long-handled scissors. No anesthesia is necessary but of course epidural anesthesia is useful. Suturing is unnecessary. Vaginal douches should follow for several days. At the end of ten days to two weeks, one will find in place of the dilated os with the rose-like eversion, a small cervix of normal size and color.

In group IV one finds the deeply infected and lacerated cervices. The entire cervix is involved. Its walls are thick and hard, its lumen open and filled with mucus or pus.

The "plicated palm" is made up of numerous elongated polypoids matted together to form an amorphous mass that obscures a chronically dilated cervix.

The tumor is covered with slime that also fills the lumen. In fact, this type of endocervicitis is invariably associated with endometritis (usually of the third degree).

The cervix, instead functioning as a barrier to the introduction of infection into the uterus (because of the size of its lumen and the infection it harbors), constitutes a perpetual menace and invites, rather than inhibits, invasion of microörganisms.

Treatment is discouraging. Caustics usually fail as well as other forms of medicinal applications. Surgery offers the only relief for this condition. The operation is thoroughly described by W. W. Williams, under the title of "Circular Amputation of the Cervix."

The prevention of infection of the cervix should begin at the time of parturition, since the cervix at this time is fully exposed to any invader that might enter the genital tract.

As a whole, veterinarians are very careless in the handling of obstetrical cases and more so, perhaps, in the treatment of retained fetal membranes. The circumstances under which we are forced to work are not conducive to cleanliness but, on the other hand, a great improvement can be made in the routine used by the most of us.

In normal parturition no interference should be permitted. But in handling cases of dystocia, abortion and retained fetal membranes, not only the uterus but also the cervix should receive attention. In the first place, while delivering a fetus, one may avoid lacerations by taking plenty of time, using sufficient lubrication and applying judicious traction.

Disinfection should begin early and be continued until health is restored. Immediately following delivery of the fetus, the cervix should be soaked with mercurochrome or acriflavine solution. Fluids that pass through the cervix from an infected uterus are highly dangerous to the former. Also fluids that are retained in the vagina are exposed to the outside and naturally become contaminated. Thus the cervix receives a continual baptism of noxious material.

Cleanliness in handling the cervix, and disinfection at the time of parturition, followed by hot vaginal douches of salt or sodium bicarbonate, aid toward preventing endocervicitis.

DISCUSSION

DR. J. A. JONES: Which speculum do you use most, Dr. Kingman, the glass or metal?

DR. KINGMAN: I made myself a speculum. I don't use the glass kind, because we broke so many of them. I asked Dr. Graham to find us a rubber tube, which he did. But he wouldn't consent to light it. So I took a flashlight bulb, fastened it in the end, and ran my wires up the wall and hooked it on to the flashlight battery.

There is a very nice nickel-plated speculum on the market that was designed by Huddleson and improved by Case.

I made two sizes: I have a little one that is introduced into heifers. I use it for diagnosis of pregnancy and for the diagnosis of cervicitis, and find it very useful. It stands rough usage and sterilization. And the tube costs less than five dollars. That is another factor with Colorado veterinarians, the financial end. I notice that doesn't apply to the West Coast. (Laughter)

DR. JONES: You make a practice of never introducing your hand into the vagina?

DR. KINGMAN: No, I don't make that a practice. Heifers are so small it becomes impossible. And, particularly, in the diagnosis of pregnancy I don't think it is a good idea to introduce your hand unless you feel it is essential. I don't think there is any particular danger associated with it, but if one can look through a well-lighted speculum, the whole section is thrown open to you much more easily than in any other way of examination. Of course the cervix is sealed in most instances. If we are confused in any way, we go ahead with further examinations.

DR. R. A. BALL: Most of these cases, as I understand them, are of fairly long standing, and what you might term neglected cases, are they not?

DR. KINGMAN: Yes.

DR. BALL: It seems to me that this is one of the most promising fields of the profession. And it also seems to me that we are making a great mistake by letting so many technical cases appear. I have been trying to specialize in this work for the past twelve years. We started our work by treating the animals every four days. We found out that it caused too much inflammation, irritation and adhesions, so that it has gradually worked along in my cases where I make examinations every two weeks. It finally developed into a system of handling herds, a system of examining the entire herd. Usually a herd that we take charge of would have ten per cent or more of the animals going to the slaughter-house for sterility within a year. In such herds we determine the true standing of the herd by examination because, oftentimes, the records have been sadly neglected.

After that first examination, three groups of cattle appear: (1) the sterile group, (2) the fresh cows, (3) and the pregnant.

On visits following the first examination these three groups automatically fall in line. The longest time that a cow goes without examination after calving is two weeks. The shortest time—maybe she calved in the morning, just before you arrive. Under such a system as that you get away from this.

It seems to me that this is the great, wide open field to the country practitioner—to quality himself to handle these things so that at the end of a year you have a higher percentage of conceptions in the herd and a satisfied client.

Naturally, the first year is trying, especially the first six months. The owner is very undecided as to whether you are doing him any good or not. Of course, keeping an exact record, a detailed report of everything you do, especially checking against the breeding dates that appear on the chart, within a very short time, as soon as these fresh cows have had time to appear on the breeding record, he sees in his record that you are really accomplishing a great deal for him.

It was reported by a prominent veterinarian in this state last week that a single repeated breeding costs a dairyman selling market milk, twenty-one dollars. That is three times the estimate that I have placed upon the value of such services. But it really is enormous, the benefit that can be derived by the dairyman along this line of work; it nets him around three to five hundred per cent on the services of the veterinarian.

DR. J. E. MCCOY: I was very interested and pleased with Dr. Kingman's paper. He brought out the subject in a clear and concise manner.

Endocervicitis is one of the common causes of sterility. I wonder if Dr. Kingman has in his mind the percentage of sterility that occurs from cervicitis as a primary cause?

DR. KINGMAN: We have no statistics along that line. I don't know that it would be possible. The subject of sterility is such a complicated one that I think one would have difficulty in saying, "I know definitely that this is the cause and the only cause of this and that case of sterility,"—if I catch your question.

I prefer to answer that in a roundabout way, by saying that I presume that in seventy-five per cent of the cases that I encounter, cervicitis is present. Dr. Rankin has called your attention to the fact that exudate escaping from the uterus would infect the uterus so commonly that the two would go together.

DR. MCCOY: That gives your attitude at least. From the medical standpoint, at least, it seems to me that when you find a cervicitis, ordinarily if you treat the cervicitis and get results, sterility will appear. We, perhaps, can deduce then that sterility is primarily caused by cervicitis in those cases.

Dr. Rankin's paper brought to my mind a question: He alluded to the use of live cultures in controlling these abortions. It seemed to me that this inference was that the results had been pretty good, from a clinical standpoint. Now, we are in the Section on General Practice here. Over in the Section on Sanitary Science we might not be able to discuss this subject, but I am wondering what Dr. Rankin's attitude is, clinically, toward the use of live cultures in the control of this trouble?

DR. RANKIN: Dr. McCoy, personally I don't think much of live cultures. I stated in my paper that those heifers that I vaccinated with live cultures aborted just as large a percentage as those that I didn't vaccinate. I used only some of the young cows, mostly heifers that might some time have aborted a calf, but I don't think much of live cultures.

I just used vaccine in some cases. That was a badly infected herd, infected with Bang's disease. It was unusually bad, around ninety per cent in a herd of probably three hundred head. I am not very strong for it myself. I haven't had much experience; I haven't used it on enough cows to draw any definite conclusions to put out that would be authentic, or anything like that.

DR. T. H. FERGUSON: I am very much interested, and have been for a long time, in this class of work. And I want to compliment the officers of this section on the nice way they have presented these two subjects.

Cervicitis is a very important condition in districts where dairying is carried on, and I presume that the same holds true in beef herds. These papers have discussed this matter very clearly and concisely. The presentation of the subject has been excellent. A lot of practical information has been brought before us and a nice picture of the more common cervicitis, or inflammation of the cervix.

I agree heartily with the plan of controlling this trouble by examining and treating cows shortly after parturition, rather than letting the cervicitis, or other trouble, develop to such an extent that it becomes chronic, and is then very difficult to treat. Take the last two degrees of cervicitis—they are difficult to treat, and unless the animal has a lot of value, or the expense is not taken into consideration, she had better be sent to the slaughter. If they are valuable, and you have a rich client, it pays a good many times to operate on them and treat them. But the ideal time to treat sterility is early. And if you have the health of the herd under your charge, and if you are making a specialty of treating sterility in cattle, get to them early. Make a practice of examining every cow in the herd shortly after parturition.

You will find a good many times, in large herds, cows that are off feed, probably two, three, four or five days after calving—they have been eating a little, but not doing so well. All at once they have stopped eating; their ears have drooped; they carry a few degrees of fever, the rumen is lazy, and you might be deceived and think that it is a case of indigestion or something. Go into those cows and make an examination of the uterus. You will find the os is contracted, and anywhere from a quart to half a gallon of fluid, and very often some gas, in the uterus. By dilating the cervix and massaging out that

fluid carefully, immediately your patient is better. Follow that up with the introduction of Lugol's solution or mineral oil. Take a gallon of mineral oil, and pour an ounce of Lugol's solution in it; shake it up and let it stand. The oil will float on the top. It will not take up all the iodin; probably half of it will settle on the bottom. Pour off the clear, nice-colored solution and you have a good, safe preparation that is reasonable in price, that you can introduce into a uterus and it will do a lot of good. It will float up and out any débris that may be in there.

If you handle a case of that kind in this way, maybe just one treatment will give that cow a nice start. Maybe it is all she needs, or if she happens to be a case more advanced, and her uterus is inert, follow that up in a day or so with another treatment. Of course, you have to judge according to the condition you find, but many of those cases are straightened out with just the one treatment.

If she has what we call a "juicy cervix", that is, if it is rather more on the order of a "snotty" cervix, the external portion real dirty, in addition to that we always flush off the external os and the vagina with a nice hot saline douche and leave the animal clean.

If we find that there is considerable irritation there and the animal has to strain considerably, we just give an epidural block between the first two vertebrae, of ten or fifteen cubic centimeters of a one per cent solution of apothesine, enough to make the tail limber. Then go at the cervix and clean it up nicely. You will see that after the effects of the anesthetic wear away, the cow will be relieved.

There is another condition that we very often find associated with cervicitis, which the essayist did not see fit to include in his paper, one that is very often associated with cervicitis—and that is salpingitis, or tubular trouble. I have met with that a great many times, and I think very often there is an intimate connection between the two.

Then there is another cause for cervicitis that you may find existing extensively in a herd, and that is the cervicitis that is caused from an infected bull. Those are nasty cases. You have a bull, and some part of his genitalia is infected with a bad pus-producing organism that causes a lot of trouble. He will serve a heifer or a cow and almost immediately she will develop intense cervicitis. A cow that is apparently clean and all right today receives a service from such a bull and in a few hours you will notice that she has a profuse discharge, and in a few days you will notice it very much—there is a lot of mucus or pus coming away from her. And every cow bred to that animal will develop that condition.

So there is another condition that you must watch—the male in the herd. Be sure, if you are having a lot of trouble with cows that are being bred to a certain bull, to make a thorough examination of that bull.

A good many times, from an external examination, you can determine whether or not he is diseased. It may be the vas deferens, the seminal vesicles of the testes. Then, again, you have to have his semen examined in order to tell. But from a clinical standpoint, be suspicious of a bull whose cows or heifers are discharging after service.

I have been mixed up in quite a lot of cases with cows apparently clean and healthy that have been sent to a high-class stock farm where there was a bull of merit, as far as breeding was concerned, and they wished to get some of that blood, and a good many times had unsatisfactory results due to the infection carried to these cows by that bull. A good many times it causes a lot of trouble and litigation; whereas, if the veterinarian in charge of the herd pays close attention to the male as well as the females, he will avoid a whole lot of trouble.

Now, the question one gentleman asked about the use of live cultures in treating abortion disease: The best you can say for live cultures is that some reports show that in some particular instances apparently they have reduced the actual number of dead calves. I believe that is the best that you can say for live-culture vaccine.

On the other hand, what you can say against it is a whole lot. I won't go into that because you probably all know more about it than I do, but from a personal standpoint I can say a lot against it.

I believe that a practicing veterinarian can do more good for the live stock industry and his clients by practicing sanitation on all of these cases than in any other way. This job is a veterinarian's job, and he should, in assuming his responsibility, put himself in a position to handle it properly. The only way he can do that is to attend these lectures, and read up on these subjects, and then get into the game in the field actively and do some work there. He has to have the actual contact, the actual experience; he has to take off his coat and learn how to work and diagnose these cases. After he gets considerable experience, it is easy work. At first it is hard work, like everything else. But you get a lot of enjoyment out of every one of these cases that you treat.

Now, the question is: What is the practicing veterinarian going to do with a client who is just having a storm of abortion breaking out in his herd? Somebody tells him to use live cultures; another tells him to use a little medicine. It is up to him. He faces the proposition. The psychological aspect of the case is sufficient to relieve that man's mind. You can't use sanitation—it doesn't appeal to him. He wants to stop his trouble. I believe that a veterinarian is justified in using a reliable bacterin in those cases, not particularly for any good that might come from it but to compete with about a thousand useless remedies that are costly, and just take a man's money for nothing.

In this case you are not selling the bacterin; you are just using it to help bring about a condition of affairs that will permit you to straighten out this herd on good, sound, sane practice. And I believe, on those grounds, that one is justified in using it.

I would like to ask Dr. Kingman to say a word or two about the relation, in his experience, of tubular trouble to cervicitis.

DR. KINGMAN: Dr. Ferguson, I haven't encountered a great deal of tubular trouble that I could recognize.

It seems to me, in the diagnosis of tubular trouble, it is dependent upon one of two factors: The first is an anatomical change of sufficient size that it may be recognized by palpation. The second is through a process of elimination, reaching the conclusion that, everything else being normal and unable to account for this condition in any other way, possibly we are dealing with salpingitis.

I wouldn't infer that we do not encounter recognizable tubular trouble commonly, but in my mind and my experience, I have had difficulty in bringing myself to a feeling that I have actually diagnosed it very commonly.

There is a matter that I think needs emphasis, relative to endometritis and its relation to Bang's disease, and that is that Bang's disease is a specific endometritis of the gravid uterus. I think that we lose sight of that a little bit. In the human we have gonorrhea as a specific inflammation of the genital mucosa. In our practice we encounter Bang's disease. Remember that abortion is endometritis of the gravid uterus due to a specific organism. That is the most common form of endometritis.

I believe that is a factor that is commonly overlooked. We try to separate endometritis from Bang's disease; that shouldn't be attempted. I think we should get to thinking of Bang's disease as the most common endometritis that is encountered.

DR. J. A. BESTAL: I would like to ask if either Dr. Kingman or Dr. Rankin has had any success, or knowledge of anybody having success, in the use of biologics in conjunction with the medical handling of these cases?

DR. KINGMAN: I have the opportunity of being associated with a school and having autogenous bacterins made whenever we feel that we want them. I have used autogenous bacterins in the treatment of endocervicitis and endometritis, and I believe that they have value; in fact, I think that one should take a swab from the cervix when a laboratory is available and check up the cause if possible. Where one is dealing with separated causes, particularly, I believe that undoubtedly we gain a great deal through the use of autogenous bacterins. I am not familiar with the use of the stock bacterin in this condition.

DR. RANKIN: I have not used enough vaccine to make any definite conclusions.

DR. W. L. BLACK: Within the last year I have had rather a peculiar experience. Our Director of Extension had a cow with a chronic case of mastitis. We had given her bacterin treatments, and had done everything we knew, and

had not helped her any. So we finally put her on a mixture made out of seaweed and fishmeal—whatever it was—and she immediately recovered. And we had worked on her a year trying to cure her. But when we put her on this she immediately recovered.

The striking thing of the whole proposition was this: That man had been troubled with chronic constipation for years. But since he has been drinking the milk from that cow he has been cured; he hasn't it now. (Laughter)

Is there or is there not a connection between that and these infections? It is rather striking to me.

DR. RANKIN: Can someone answer Dr. Black's question?

DR. G. S. GLOVER: This is rather new to me. You spoke about fishmeal which we, in this section know as manaca. I became interested a month or two ago in finding out what this mineral idea was. We had men coming to see us, salesmen telling us that all we needed to do was to put these cows on this mineral, or that mineral, and all our troubles would fade away. So I proceeded to find out from our herd, taking the daily ration of the milking strings. In this herd that I have been speaking of, we have twelve hundred animals. I direct the service. I find that on the Coast here, where we are feeding alfalfa, that we get a great excess of calcium as balanced against the phosphate. The other minerals I balance. I wish I had the figures with me, but I haven't them, of the other minerals; but the chief thing was calcium and phosphate.

Then I undertook to balance this against this idea of feeding two per cent mineral, paying anywhere from eighty to one hundred and fifty dollars a ton. And I found that in a pound and a quarter of bran we feed—I am stating these figures from memory, and it is damned poor (Laughter)—thirty-six times more phosphate than we would in the mineral of kelp at two per cent of the ration. We feed something like eighty-six times more calcium in ten pounds of alfalfa a day than we would in kelp at two per cent of the ration.

Taking the milk—they tell you, "Well, it goes out in the milk. The good cows lack mineral." You look at them and they look fine. I found what the required mineral was for a cow giving forty pounds of milk. There is no mineral that will put back the mineral taken out in the milk for a forty-pound cow if you give that cow two per cent of the ration.

I studied that from the dicalcium phosphate which, I believe, is highest in calcium and phosphate of any known product today.

I do not say that minerals are not needed. Certainly on the Coast here we are feeding a lot of minerals in our feed. But the addition of two per cent of the ration, judging from my conclusions, doesn't do any great deal of good. It doesn't put as much back into the milk as is taken out if the cow is giving forty pounds.

There may be some catalytic action in dicalcium phosphate if it is acid-soluble. If there is any value—and I believe that I drew this in my conclusions—in kelp or kelp and fishmeal, it is not due to the mineral content, but is due to something else.

DR. BESTAL: Dr. Ferguson mentioned in his talk a few minutes ago something about the dilation of the os for the purpose of introducing different materials into the uterus. I wish to ask him what is his modus operandi in the dilating of the os, and what the possibilities of damage are to the tissues in the operation.

DR. FERGUSON: The cases I spoke of were cases following parturition, at which time this trouble developed. At that time the os was partly dilated and it was easy to do the dilating with the fingers. In fact, you could easily get a finger or two in. It was just a question of slipping in and getting your fingers into the os and pulling it back with the hand in the vagina and reaching into the rectum and massaging the débris out of the uterus, which consisted almost always of a fetid, gaseous fluid. That is what you will find in most of those cases, that the os hasn't completely contracted into a closed condition.

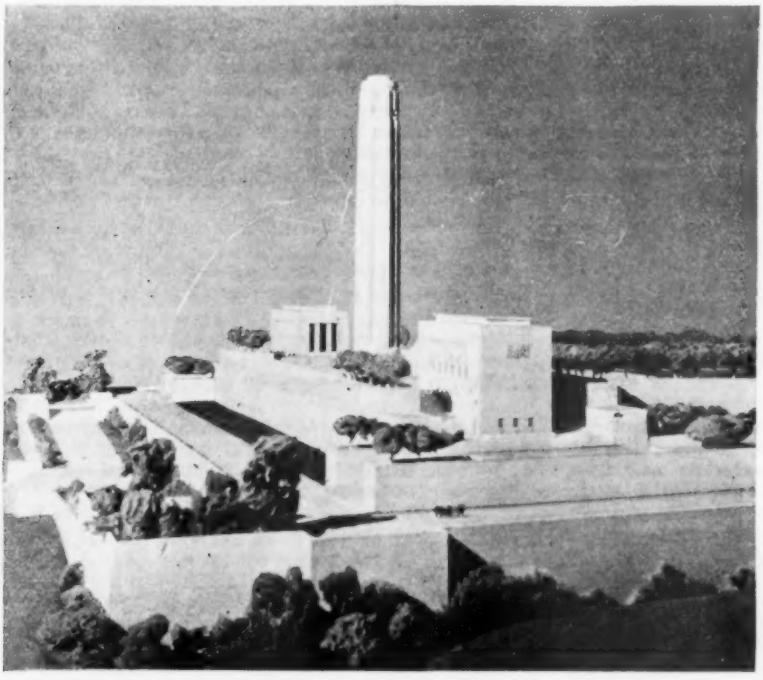
Of course, speaking of dilating an os when it is closed—that is a different proposition. But you would have no trouble in these cases. The cases in

which I recommend the above treatment are cases that show up thirty, forty-eight or seventy-two hours, maybe ninety-six hours, after calving; cows that have come in, in most instances, all right and apparently clean. In a good many of such cases there is nothing apparently wrong up to the time of the dullness and inappetence. In other instances you will find some indication of Bang's disease, but a great many cases occur in cows that calve and clean apparently normally.

## Veterinary Officers to Meet at Kansas City

The program for the meeting of the army veterinary officers to be held in connection with the A.V.M.A. meeting in Kansas City is practically completed. There will be five papers, two by regular army officers and two by reserve officers. General Sir John Moore, who was chief of the British Army Veterinary Service in France, will send a paper. General Moore has spent considerable time in the United States, as he was stationed at Lathrop, Mo., where the British had a remount depot during the South African war.

N. S. M.



LIBERTY MEMORIAL, KANSAS CITY
Hall of Memories and Museum contain treasures relating to the World War. Located just south of Union Station.

# STUDIES IN THE PATHOLOGY OF AVIAN COCCIDIOSIS*

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## INTRODUCTION

Coccidiosis in chickens is a disease which is steadily gaining in prevalence and importance. It is no longer considered to be a menace to young stock only, as we now know that fully grown birds are susceptible and may suffer rather severely from this malady. Hundreds of chickens, young and old, are sent to this laboratory annually to be examined for positive evidence as to the presence of coccidia in diseased birds. The history of the disease outbreaks, submitted with the various consignments, together with the gross and microscopic findings seemed to indicate that lesions, pathognomonic of coccidiosis, usually occur during the first two weeks or so of the disease but may later change to such an extent that it is difficult to make a diagnosis merely on the basis of pathological changes, *i. e.*, when one is dealing with the disease as it occurs in the small intestines. Moreover, it was noted that chickens, sent in during the early stages of the disease, invariably showed numerous coccidia in the intestinal contents or scrapings from the mucous membrane, while birds that had been sick long enough to show such symptoms as paralysis or extreme weakness often appeared to be entirely free from these parasites.

This study was undertaken in the hope that enough might be learned concerning the pathology of coccidiosis to enable us to diagnose this disease more exactly in its chronic forms.

## HISTORICAL

There is not a great deal of literature available covering the pathology of avian coccidiosis, excepting, of course, the usual descriptions of the gross lesions of the common form of the disease as it affects chicks. As most books and bulletins on poultry diseases treat this phase of coccidiosis quite well, no further mention of it will be made here. The chronic form or the type that affects mostly the small intestines is less fully described and very

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few publications indeed contain anything concerning microscopic lesions.

Eckardt[1] states that in subacute or chronic cases the intestinal mucous membrane is reddened in spots, coated with tenacious mucus and as though dusted with flour. Such dust-like points occasionally occur also in the liver; they are then also due to coccidia. Hadley[2] describes quite fully what he calls subepithelial infection. He found "merozoite cysts" adjacent to the base of the crypts and in the core of the villi and some times they were packed in the cores to the exclusion of nearly all other cell structures. In such cases they crowded closely on the basement membrane and were often packed solidly along the muscular wall or in the muscularis mucosae.

Ward and Gallagher[3] have merely this to say about the anatomical changes in the small intestines: "The lining membrane is deeply congested or hemorrhagic." Hutyra and Marek[4] mention intestinal catarrh or enteritis with patchy desquamation of epithelium. They suggest that the sudden death may be due to secondary infection. Reinhardt,[5] besides describing the anatomical changes occurring in the acute form, mentions the occurrence of diphtheritic membranes and small pin-head-sized grayish foci in the mucous membrane of chickens affected with subacute or chronic coccidiosis. He says that those foci are nothing else but immense masses of macro- and microgametocytes in the epithelium of the gland tubules.

Joest[6] found coccidia in the epithelium of the villi and the intestinal glands, with desquamation of epithelium. In places the epithelium was completely lacking. The connective tissue of the propria was infiltrated with leucocytes. Otte's[7] description of the intestinal lesions of coccidiosis is very similar to those by Eckardt and Reinhardt. He states that in the liver of older birds one can notice light spots. Krijgsman (v. Heelsbergen)[8] describes the pathological anatomy of peracute, acute, subacute and chronic forms, mentioning very briefly some microscopic lesions. The changes noted do not differ from those already referred to. He mentions grayish white foci in the liver as being lesions of coccidiosis but adds that it is not sure that these lesions have not been confused with those of blackhead.

Tyzzer[9] has recently published his results of some very extensive studies on the nature of coccidiosis in gallinacious birds. He describes four species occurring in chickens:

1. *Eimeria tenella*, the cause of cecal, acute or hemorrhagic coccidiosis. Epithelial cells infected with this parasite become phagocytic and actively infiltrate underlying tissue, hence the apparent subepithelial infection. This parasite may also infect the lower part of the small intestines. There is sloughing of mucous membrane five days after infection. There is more or less eosinophilia in the region of glands infected with first generation merozoites. He also finds lymphoid and plasma-cell infiltration with increase of connective tissue in these areas.
2. *Eimeria mitis* (n. s.) occurs mostly in the upper portion of the small intestines, does not tend to occur in swarms but tends to penetrate beneath the nucleus of an infected cell. Little damage is done by this species.
3. *Eimeria acervulina*, associated with chronic coccidiosis and perhaps the most common of the four species, found mostly in the upper part of the small intestine, tends to mass in limited areas, producing gray spots in the mucous membrane. This species merely penetrates the cuticular layer of the epithelial cell, causing patchy desquamation of epithelium.
4. *Eimeria maxima* may be distributed throughout the entire length of the small intestines, the greatest numbers being found in the middle portion. The mucous membrane becomes swollen, covered with dirty grayish exudate and may show some hemorrhage. The pathological changes are associated with the sexual portion of the life cycle. The gametes are large and develop deep in tissues. The asexual forms are small and are situated superficially. He states that deficiency diseases may develop as a result of chronic coccidiosis. No evidence was found to show that coccidia produce lesions in the liver. Kaupp's[10] description of the postmortem findings agrees with those of the other workers whose work has been reviewed.

## Experimental

*Source of material:* One part of the material consisted of chicks, growing stock or adult birds sent to this laboratory for examination. The other part consisted of experimentally infected birds. Such birds were kept under observation for some time to see if they showed any evidence of being infected with coccidia. Their droppings were examined for the presence of oöcysts and the general appearance of each bird was carefully noted. If symptoms of disease were observed or coccidia found in the droppings, the bird concerned was not used.

*Methods:* The coccidia were sporulated in wide glass dishes on the bottom of which were placed two layers of filter paper. A two per cent solution of potassium dichromate was used to keep the "culture" moist. Prior to feeding the "culture" to chickens it was either washed with 0.6 per cent NaCl solution or it was diluted with water and grain mash. This was done either to remove the potassium dichromate or to dilute it to such an extent that it would not be likely to injure the sporozoites. This precaution may be entirely unnecessary as some birds that were fed cultures containing considerable amounts of the dichromate solution promptly developed coccidiosis.

The "cultures" were fed either in grain mash or with 10-cc volumetric pipettes inserted into the esophagus. The "cultures" used were sufficiently rich in organisms so that every microscopic

field (high power, dry) showed from one to five or six coccidia. If it was desired that reinfection should take place, the birds were kept in cages with solid bottoms and no precaution was taken to keep the cages, feed and water clean. When reinfection was not desired, wire bottoms were placed so that contamination was not likely to take place.

Since I was mainly interested in studying chronic coccidiosis, most of the birds were given several moderate doses or they were kept in contaminated pens or cages for several weeks if necessary.

The tissues taken for sectioning were fixed in Zenker's solution and stained with eosin and methylene blue.

## Paralysis Associated with Coccidiosis

In the summer and fall of 1924, 1925 and 1926, rather extensive outbreaks of leg weakness and paralysis occurred in a flock over which we had close supervision. The birds affected were kept on a piece of ground that had been used as a poultry range for many years. These birds showed rather heavy tapeworm infestation (*Davainea proglottina* and *cesticillus*), together with coccidiosis. A portion of this flock, kept on a range lying on the side of a hill, the soil of which, being of a sandy or even gravelly consistency, remained free from paralysis and coccidiosis. Until this fact was noticed, the caretakers had thought the lameness and paralysis to be due to vitamin and mineral deficiency. That the feed could have nothing to do with this ailment was very evident, since all birds in this flock received the same ration.

In order to study the possible relation between leg weakness (paralysis) and coccidiosis, twelve normal birds (White Leghorns) were placed in a pen, August 31, 1926, and fed sporulated coccidia, August 31, September 1, 6, 17, October 10 and November 27. On September 29, one cockerel was found to be very lame after having shown incoördination for two or three days. On October 4, this bird was completely paralyzed and was killed. The following is the autopsy record of this bird (308):

### Gross Lesions

*Liver:* Several clusters of grayish spots and a few such foci scattered over the entire surface

*Intestines:* Hemorrhagic enteritis with numerous grayish spots resembling minute necrotic foci also visible through serous membrane. Much slimy material throughout intestinal canal anterior to the ceca.

*Ceca:* Several petechial hemorrhages visible through serous coat. Contents are caseous, grayish to grayish brown and red in areas. The cheesy mass adhering to the walls of the ceca leaves a raw surface when removed.

MICROSCOPIC

*Scrapings of mucous membrane:* Innumerable small coccidia in various stages, oöcysts and intracellular elements.
*Sciatic nerves:* Normal.
*Intestines (sections):* Several large foci of small coccidia in epithelium and subepithelial tissue. The coccidia have invaded the tissues down to the fundus of the crypts in some places. There are hemorrhages and sloughing of the mucous membrane.
*Liver (sections):* Numerous areas of perivascular infiltration with undifferentiated mesenchymal cells.
*Nerves:* Normal.

On October 28, 1926, another cockerel (bird 386) was found very emaciatd and completely paralyzed. The autopsy record is as follows:

GROSS LESIONS

*Pericardium:* Much serous fluid.
*Spleen:* Very small.
*Gall-bladder:* Distended.
*Intestines:* Numerous grayish spots in duodenal mucosa. There is some evidence of sloughing of the mucous membrane in some areas.
*Ceca:* Normal in appearance.
*Nerves:* Normal.

MICROSCOPIC

*Scrapings of mucous membrane:* Innumerable very small coccidia.
*Intestines (sections):* Several extensive areas of epithelial and subepithelial infection with coccidia. There is marked destruction of epithelium and displacement of subepithelial tissues in these areas. Some villi seem completely broken down with only a little muscular tissue remaining. There seems to be no cell reaction in the infected areas.
*Nerves:* No cellular infiltration.

On October 29, 1926, a third cockerel (bird 390) was found in this pen showing the following symptoms: excitability, anemia, emaciation and some diarrhea (not bloody). This bird was killed and the autopsy record follows:

GROSS LESIONS

*Heart:* Very flabby.
*Liver:* A few small gray spots.
*Intestines:* Pale with small grayish spots in duodenal mucosa. Several round worms.
*Ceca:* Normal.

MICROSCOPIC

*Scrapings from ceca and duodenum:* A number of coccidia.
*Liver:* There is a moderate perivascular infiltration with undifferentiated mesenchymal cells. Some of the capillaries are injected.
*Intestines:* Marked areas of infiltration with undifferentiated mesemchymal cells in the stroma and muscularis mucosae. The epithelium is well preserved except over the tips of the villi where there appears to have been some mechanical destruction. There is congestion of the capillaries in the deeper portions of the mucous membrane. In some sections the greater portion of the villi is missing. This may be due to mechanical influences due to the handling of the tissues, but might also be due to a recent coccidiosis infection. No coccidia were recognized in the sections studied.
*Nerves:* Normal.

Later, three more birds developed chronic coccidiosis, showing anemia and emaciation. About the middle of November, 1926,

the remaining six birds were moved to a clean pen with concrete floor, and a few days later another bird was found showing leg weakness. A postmortem examination revealed duodenal coccidiosis. None of the other birds developed any symptoms of disease and were disposed of late in November. A number of birds (more than fifty) were kept in three pens adjacent to the one used for this experiment for more than one year and not a single case of lameness or paralysis had developed among them. All these birds received the same kind of feed.

Following the publication of a preliminary report[11] on this experiment, it was suggested to me that the lameness and paralysis observed in coccidiosis might be due to nothing but general weakness. However, further studies have shown that leg weakness seems to be associated more often with duodenal coccidiosis

TABLE I—*Incidence of leg weakness or paralysis in 326 consignments of coccidiosis-infected chickens*

| | | With Leg Weakness or Paralysis | Without Leg Weakness or Paralysis |
|---|---|---|---|
| Duodenal Coccidiosis (113 Consignments) | Consignments | 80 | 33 |
| | Percentage | 70.8 | 29.2 |
| Cecal Coccidiosis (174 Consignments) | Consignments | 5 | 169 |
| | Percentage | 2.87 | 97.13 |
| Cecal and Duodenal Coccidiosis (35 Consignments) | Consignments | 19 | 16 |
| | Percentage | 54.29 | 45.71 |

*4 Consignments (3.54 per cent) showed blindness.

than with cecal coccidiosis. Therefore, since cecal coccidiosis is usually the more severe type, one can hardly feel justified in attributing leg weakness or paralysis merely to general weakness. Table I shows the incidence of lameness or paralysis in 326 consignments of coccidiosis-infected chickens (young and adults) sent in for examination over a period of three years.

While it is true that these data do not prove that duodenal coccidiosis is the cause of leg weakness and paralysis, they at least suggest a significant trend.

Paralysis has been observed in chicks as young as three weeks. An outbreak of paralysis occurred in a large number of seven-week-old chicks on May 21, 1927. The owner brought in nine

paralyzed chicks (some White Leghorns, some Barred Rocks) for examination and the history as given by him was as follows:

> Had done well until this week. Only a few pens affected. There is no bowel trouble. The first symptoms are dizziness and twisting of the neck. Some throw head right back. Toes are cramped in some of them. In others the first thing noted is that they are down completely paralyzed. Chicks ultimately die and twenty-five have died so far.

The owner has examined several chicks and found no signs of cecal coccidiosis. All nine chicks showed severe catarrhal duodenitis and scrapings from the mucosa of eight of them revealed innumerable small coccidia. The ceca seemed normal. Coccidiosis control measures were suggested and no more similar trouble has been encountered.

On June 16, 1927, two pigeons and two chicks were brought to the laboratory. The history, as given by the owner, was as follows:

> Leg weakness and diarrhea occur in some chicks and pigeons. The latter also lose control of wings. Several of the pigeons had become paralyzed without showing diarrhea. A few pigeons "went light," lost appetite and became droopy.

On postmortem examination one pigeon showed numerous small white spots in the liver. A mixed coccidiosis infection (small and large coccidia) was found in the chicks. In the pigeons there was duodenal catarrh and scrapings from the duodenal mucosa showed numerous small coccidia similar to those found in the chicks.

### Deficiency Disease Secondary to Coccidiosis

Rickets has been observed in growing chickens affected with coccidiosis. Not infrequently such birds have received cod-liver oil and lime in addition to a well-balanced, grain and mash ration. Some of them have also been out-doors and have had access to green feed and sunlight. In a case report[12] on this subject, it was suggested that the coccidiosis infection might so interfere with the function of the mucous membrane as to make it impossible for these chicks to utilize their food properly.

### Coccidiosis in Sparrows

On June 22, 1927, seven chicks, about six weeks old, were brought to the laboratory by a member of the college staff who was carrying on some nutrition experiments. These chicks were extremely pale and had acute, hemorrhagic cecal coccidiosis. The contents of the ceca in every instance consisted of liquid and partly hemolyzed blood. As these chicks were kept in clean quarters on the second floor of a college building, the question

was asked: "How do coccidia get up there, can it be that sparrows carry them from the poultry yard?" Cole and Hadley[13] and Kaupp[14] have suggested that coccidia may be carried by sparrows. Smith and Smillie[15] found this not to be the case. In order to see what local conditions might reveal regarding this problem, forty-nine sparrows were caught in the poultry-yards of the College and examined for the presence of coccidiosis. Twenty-two (44.89 per cent) of them had coccidia of the genus Isospera. These coccidia were found in the posterior part of the intestines, the rectum and cloaca. Numerous white spots were found in the mucous membrane of the cloaca. These spots proved to be colonies of coccidia. None of the sparrows revealed any coccidia of the genus Eimeria.

## Relationship of Coccidiosis to Leg Weakness, Paralysis, Torticollis and Blindness

In a further attempt to prove or disprove the relation between chronic coccidiosis and such manifestations as leg weakness, paralysis, torticollis and blindness, a study was carried out on twenty-two birds showing one or more of these symptoms. The birds were nearly fully grown or adults, obtained from different sources. Some of the most interesting ones of the case reports from this work follow:

### Case 260

*Subject:* White Leghorn hen, Hartford.
*Symptoms:* Emaciation, cyanotic comb.

### Gross Lesions

*Liver:* Numerous small grayish spots of the size of a pinhead.
*Heart:* A few petechial hemorrhages.
*Intestines:* Numerous tapeworms (*D. cesticillus*), round and cecum worms. No record of coccidiosis lesions or coccidia in intestines.

### Microscopic

No record of coccidia in scrapings.
*Intestines (sections):* A considerable number of small and a few medium-sized coccidia in subepithelial tissue. Epithelium is broken up as if by mechanical means. In one section there is a very large focus of coccidia covering an area too large to be seen at one time with the high-power objective (No. 6, Leitz). (See figure 1). This focus is located deeply in the subepithelial tissue, in the middle portion of a villus. The tissues surrounding this focus seem not to have been injured. In the area of the focus, however, the coccidia have completely replaced the normal tissue. A few areas, nearly as large as the focus just mentioned, are found in other villi, in which there are no coccidia but there are open spaces partly filled with tissue débris suggesting that this may be the result of focal coccidisosis infection, the coccidia having disappeared. The location of these areas corresponds exactly to that of the colonies of coccidia. Near some of these areas coccidia, which seemed to be in the gametocyte stage, were found.
*Liver:* Numerous areas of perivascular, undifferentiated, mesenchymal cell infiltration (figure 11). These are fairly well distributed throughout the liver. Near the posterior border of one lobe, immediately under the surface, are con-

glomerations of numerous small areas of this sort. These areas are often observed in chickens. Bacterial cultures from livers showing such lesions are usually negative. *Escherichia coli* and occasionally *Salmonella pullorum* have been isolated.

CASE 271

*Subject:* White Leghorn.

*Symptoms:* Paralysis.

GROSS LESIONS

Sciatic nerve of left leg enlarged; slight enlargement of nerve in right leg.

MICROSCOPIC

No record of coccidia or parasites in intestinal contents.

*Nerves (sections):* Moderate diffuse and focal undifferentiated mesenchymal cell infiltration. A few of these cells are found also throughout the sections. There is marked degeneration of the nerve fibres, medullary substance and the axis cylinder. The picture corresponds well with the description of Pappenheimer.[16,17] In areas there is wide separation of nerve bres, partial and complete collapse of myelin sheaths and vacuolization.

*Intestines:* Considerable breaking down of mucous membrane. In one such area was found one schizont. The epithelium in this area is intact but the subepithelial tissue is broken up as if by mechanical means.

CASE 294

*Subject:* White Wyandotte.

*Symptoms:* Leg weakness.

*Biological tests:* Tuberculin test, negative. Agglutination test for pullorum disease, negative.

GROSS LESIONS

*Liver:* Very pale, with a few grayish specks near the border.

*Intestines:* A few small grayish spots in mucous membrane; also a few small hemorrhages.

MICROSCOPIC

Scrapings showed no coccidia in this bird but from another one from the same source coccidia were obtained. There were some small tapeworms.

*Liver (sections):* There is moderate, perivascular, undifferentiated, mesenchymal cell infiltration, extreme hydropic degeneration and retention of embryonic characteristics (gland-like tubules).

*Intestines:* Coccidia in considerable numbers have penetrated down to the muscularis mucosae. The mucous membrane is broken up in the area surrounding the coccidial focus. This has given rise to a hemorrhage. One large focus of coccidia was found in the interglandular tissue. There is the usual destruction of superficial parts of the mucous membrane. In one section showing a focus of coccidia near the muscularis mucosae the latter is broken up and there is marked, undifferentiated, mesenchymal cell infiltration in and around this focus (figures 2 and 3).

*Nerves:* Very slight, if any, undifferentiated, mesenchymal cell infiltration of the nerves. There seems to be some collapse of some of the myelin sheaths and there is also some marked vacuolization. The fibres are spread apart in some areas and in others they have disappeared.

The data obtained from the study of these birds show that nineteen of the twenty-one (86.36 per cent) harbored coccidia. Enteritis was found in all but one bird. Duodenitis, with no appreciable inflammation of the other parts of the intestines, was found in seventeen of the birds (77.27 per cent). These data are in themselves not convincing one way or the other, because of the wide prevalence of coccidia in poultry plants.

### Immune Birds or Coccidia of Low Virulence

On August 1, 1928, twenty-five normal, fully grown, young White Leghorn chickens were fed sporulated coccidia. The "culture" used consisted mostly of what was termed medium-sized coccidia. This experiment was undertaken for the purpose of showing, if possible, progressive lesions and repair processes. Two birds were killed and autopsied at 2- to 4-day intervals.

The irregular occurrence of coccidia in these chickens makes it very difficult to say whether the lesions observed in the various organs were due to coccidiosis. The intestinal changes resembled very closely those found in birds suffering from typical attacks of this disease. Coccidia were found on the 4th, 6th, 9th, 11th, 14th, 18th, 19th and 35th days after infection and, very unexpectedly, the heaviest infection was found in bird 25, which was autopsied 35 days after infection.

All of these birds were kept in wire-bottom cages (¾-inch mesh) and the feed and drinking vessels were placed as high as possible without depriving the birds of a chance to eat and drink freely, in order to guard against contamination of water and feed with droppings. If the coccidiosis infection is truly self-limiting, as claimed by Johnson,[18] and by Tyzzer in the publication already referred to, and as has been taken for granted by myself, these birds must have picked up coccidia from the hardware cloth, thus becoming reinfected. Bird 24 showed a single lesion resembling unmistakably an actinomycosis lesion in the stroma mucosae of the small intestines.

The symptoms and lesions were not so pronounced as had been hoped for. Whether this was due to immunity on the part of the birds or to a lack of virulence on the part of the culture used is difficult to say. Johnson[18] and Tyzzer[9] have shown that chickens will develop immunity to coccidiosis. Tyzzer[9] has also shown that the different species of coccidia vary in pathogenicity, the *Eimeria mitis* being practically non-pathogenic. At the time this work was done, I had no knowledge of Tyzzer's work and while I felt certain of having observed at least three different kinds of coccidia, undoubtedly *E. tenella*, *E. acervulina* and *E. maxima*, and perhaps also *E. mitis*, a coccidium which has been referred to in my notes as a strikingly round form, I had no information as to their immunological relationship. It may be of interest to note here that the birds used came from a flock where coccidiosis had been prevalent for some time. In 1924 and

1925, very severe losses were experienced in this flock due to so-called range paralysis. Since that time powdered milk feeding, as recommended by Beach and Davis,[19] together with other sanitary measures, has been employed for the control of coccidiosis and for five years losses due to paralysis have been conspicuously absent.

Some of the most interesting case reports from this experiment follow:

BIRD 14

*History:* Fed coccidia, August 1, 1928. Killed and autopsied, August 18, 1928.

*Symptoms:* Emaciated, lame on right leg.

GROSS LESIONS

*Liver:* Congested and mottled.
*Intestines:* Small, pinpoint, red and grayish foci.
*Sciatic nerves:* No visible changes.
*Heart:* Slightly enlarged and somewhat flabby.

MICROSCOPIC

*Liver:* A few necrotic foci and some perivascular infiltration with undifferentiated mesenchymal cells.

*Intestines:* There are areas of infiltration with undifferentiated mesenchymal cells in the stroma mucosae. Some of these areas show no coccidia, but others show a number of what appear to be schizonts in the middle of the infiltration areas and in others the middle portion consists of tissue débris. Numerous red blood cells (hemorrhage) and around the borders of the area are numerous schizonts (figure 6). One section shows a number of coccidia in the upper part of the villi.

BIRD 25

*History:* Fed coccidia, August 1, 1928. Killed and autopsied, September 4, 1928.

*Symptoms:* Appears normal.

GROSS LESIONS

*Heart:* Somewhat flabby.
*Liver:* Small, otherwise normal in appearance.
*Intestines:* No visible pathological changes.
*Sciatic nerves:* Normal.

MICROSCOPIC

*Liver:* Congestion and some hemorrhagic areas. There are also areas of infiltration with undifferentiated mesenchymal cells.

*Intestines:* There is desquamation of epithelium over the tips of the villi. In the epithelium of the tips of others there are swarms of coccidia. They are located below the nuclei for the most part (figure 5). A few are located superficially and others are seemingly in the subepithelial tissue. In this section one can recognize trophozoites, a few schizonts and many gametes.

It may seem unwise to have used birds coming from coccidiosis-infected stock for such experiments, but it must be remembered that coccidia are considered nearly ubiquitous and that until very recently such a thing as immunity to coccidiosis was not seriously thought of.

## COCCIDIOSIS IN CHICKS

In the summer and fall of 1929, a study was made of gross and microscopic lesions found in twenty-five chicks showing various

stages of coccidiosis. A description of the lesions found in chicks 10, 17 and 19 follows. The other chicks showed nothing of special interest that was not found in these three chicks.

### Chick 10

*Symptoms:* Anemic and droopy. Autopsied, July 6, 1929.

#### Gross Lesions

*Small intestines:* Scattered petechiae.
*Ceca:* Distended with gas but no lesions of coccidiosis.

#### Microscopic Lesions

*Small intestines:* Considerable patchy desquamation of epithelium. There are considerable numbers of coccidia in and just below the epithelium of the villi. These coccidia are rather evenly scattered. The gland epithelium is entirely free from coccidia. Most of the coccidia are found near the tips of the villi, only a few having reached down to within one-fifth to one-third of the distance from the bottom of the crypt to the free end of the villus. A considerable number of trophozoites were found varying considerably in size (11 to 14 microns in diameter). The smaller ones stain rather uniformly blue, while the larger ones show a rather distinct pinkish nucleus. Some of them are situated above and some below the nuclei of the epithelial cells. The schizonts, varying in size from 26 x 28 microns to 31.4 x 52.9 microns, were located below the epithelium. There is no cell reaction in the infected areas. In the stroma mucosae there is infiltration with undifferentiated mesenchymal cells and some indication of proliferation of glandular epithelium.

### Chick 17

*Symptoms:* Too weak to stand up. Autopsied, July 12, 1929.

#### Gross Lesions

*Ceca:* Typical coccidiosis lesions.

#### Microscopic Changes

*Small imtestines:* Many macrogametocytes staining red and several colonies of schizonts staining bluish were found in subepithelial tissues toward the base of the villi. The schizonts vary considerably in size and shape. Some look almost round, others are ovoid, one is 31.4 x 37.1, one is 37.1 x 53.4 and another 31.4 x 51.48 microns. In another portion of the same section there is a large colony of schizonts (stained bluish). They are in the stroma mucosae (figure 6). This lesion is too large to be seen at one time using a No. 6 objective, Leitz). There is marked infiltration with undifferentiated mesenchymal cells. Considerable hemorrhage and gland tubules are either destroyed or displaced. The schizonts, twenty-six in all, are situated around the periphery of the lesion. In an almost adjacent area of similar size and outline, also in the stroma mucosae, there is one mature schizont, in the neighborhood of which are three open vacuoles of the size of this schizont and several smaller ones, indicating that they represent spaces out of which schizonts have disappeared. Several merozoites, about 17 x 28 microns long, are found in this lesion, together with much tissue débris and large numbers of plasma cells. The cell infiltration extends through the muscularis mucosae and at one point, at which the fibres of the muscularis mucosae are completely destroyed, for a considerable distance into the muscular coat. Only a few plasma and undifferentiated mesenchymal cells and small connective tissue fibres occupy this otherwise open space. Oöcysts are found here and there throughout the villi in the subepithelial tissues and in other places open spaces as large as or smaller than oöcysts are found. These probably represent spaces out of which the cysts have passed. In many places the epithelium seems to be closing in from the sides.

*Ceca:* There is extensive sloughing of mucous membrane, the villous portion being almost entirely destroyed. Many glands show oöcysts in the lumen (figure 9). In the gland epithelium there are a few trophozoites and numerous vacuoles which very likely represent spaces evacuated by oöcysts. Numerous oöcysts are found in the interglandular tissues where there is also marked

infiltration with undifferentiated mesenchymal cells in many quite extensive areas. In some of these areas there are also hemorrhages. The contents consist of tissue débris, necrotic material and innumerable oöcysts.

BIRD 19

*Symptoms:* Droopy and slightly cyanotic. Legs seemed stiffened. Autopsied July 19, 1929.

MACROSCOPIC LESIONS

*Ceca:* Typical coccidiosis lesions.

MICROSCOPIC CHANGES

*Liver:* Areas of perivascular infiltration with undifferentiated mesenchymal cells. Congestion.

*Ceca:* The epithelium of the villi shows large swarms of developing forms of coccidia (figure 4). They are so numerous that they have caused extensive desquamation of epithelium. Almost every gland tubule shows immense numbers of coccidia in the epithelium (figure 10), mostly trophozoites and schizonts. In the interglandular tissues are large and smaller schizonts and numerous oöcysts (figure 8). There are some fairly large hemorrhagic areas. In one microscopic field (No. 6 objective, Leitz) three gland tubules were found in which there were so many coccidia that not a single intact epithelial cell could be detected. Throughout this section several tubules were found in a similar stage of destruction. The liberation of the coccidia from the gland epithelium takes place into the surrounding tissue and not into the lumen of the gland unless to a far lesser degree. Most of the schizonts present are nearly round and about 13.5 microns in diameter. Thus they are most likely of the third generation. The cecal contents consist of tissue débris, innumerable oöcysts, blood and necrotic material.

## DISCUSSION

In young chicks coccidia most frequently infect the ceca and sometimes the lower part of the small intestines. The result of such an infection may be fatal hemorrhage, due to the development of immense numbers of coccidia in the cecal mucous membrane causing such extensive destruction of tissues as to bring about hemorrhage through the denuded and disintegrated mucosa. As a consequence chicks may die without premonitory symptoms. In such cases a postmortem examination will show numerous petechial hemorrhages, visible through the serous membrane in the ceca, and quite often also in the lower third or half of the small intestines. The contents of the ceca will be found to consist of liquid or semi-liquid, partly hemolyzed blood.

Less acute attacks generally manifest themselves with paleness, chills (crowding), ruffled feathers, drooping wings, general droopiness and weakness. Bloody, blood-stained or brownish droppings are almost sure signs of coccidiosis. Chicks so affected will die a few hours or days after showing the first signs of disease. A postmortem examination will reveal cheesy, more or less blood-stained masses in the ceca. These cheesy masses adhere more or less to the cecal wall and leave a raw surface when removed. The lower part of the small intestines usually shows hemorrhagic enteritis, and catarrhal inflammation may be present in the upper part of the intestinal tract.

Young chicks affected with pullorum disease often have white or yellowish white, smooth, cheesy "plugs" in the ceca. These "plugs" are never mixed with appreciable quantities of blood, do not adhere to the cecal walls nor leave a raw surface when removed.

Chicks six weeks of age or older not infrequently have duodenal coccidiosis, *i. e.*, they may have a rather severe duodenal infection with less involvement of the lower part of the small intestines while the ceca are only very slightly or not at all affected. Such chicks show general unthriftiness, the appetite may be increased or decreased and, as is the case with most chicks affected with subacute or chronic coccidiosis, they may show increased thirst. Leg weakness and paralysis are rather common symptoms and not infrequently the birds look alert and quite well with the exception of the inability to use their legs. The appetite may be good for some time after paralysis has set in. Recovery does not take place but the chicks may live for many days with such an affliction.

In less severe cases all one may notice is a retardation of growth and general unthriftiness, with more or less anemia. Such manifestations are undoubtedly due to the destruction of epithelium and mucous membrane which has taken place in the small intestines resulting in increased permeability to secondary infection and toxins as well as decreased power of digestion and assimilation, or it may be due to absorption of toxic material from necrotic material in the alimentary canal. Most birds that are affected in this way fail to show involvement of the ceca. A study of the microscopic changes of coccidiosis of the small intestines showed that desquamation of epithelium, destruction of glands, and sloughing of the mucous membrane may be too extensive to allow complete repair to take place. This may account for the fact that apparently healthy chickens fail to put on flesh even when fed in the best way possible.

Duodenal coccidiosis affects fully grown birds as well as growing stock. It has been observed in fowls eighteen months old. The symptoms manifested are general unthriftiness, loss of flesh, more or less anemia or a peculiar pale, pinkish color of the comb. Leg weakness, paralysis, incoördination of movement and increased excitability may also be observed. More or less diarrhea may be present in young and adult birds affected with duodenal coccidiosis, but this is not always a conspicuous symptom.

Very seldom do we see blood in the droppings of chicks or adults so affected. Slimy droppings are noticed more often.

A postmortem examination of birds killed in the early stages of the disease will usually show more or less thickening of the intestinal wall, mostly in the duodenum but also once in a while throughout the small intestines. Now and then the middle portion will show the most marked swelling and, in rather exceptional cases, the lower part. Quite distinct, grayish spots and hemorrhages are often observed through the serous coat. The grayish spots are deep-seated colonies of coccidia. The mucous membrane is swollen, sometimes with gray spots or transverse small grayish streaks or "flakes," representing colonies of coccidia in the epithelium of the villi, and petechial or more or less diffuse hemorrhagic areas. Slimy, dirty exudate is common. Ulcers may occur and diphtheritic membranes also have been observed.

If the bird has been ill for about two weeks before the examination, the intestines will appear thin or shrunken, empty, and inflamed. There will also be evidence of sloughing of the mucous membrane. Coccidia are usually easily found except in the cases in which birds have been sick for about two weeks and show contraction of the intestines. An examination of the intestinal or cecal contents may fail to reveal coccidia, while scrapings taken more or less deeply in the mucous membrane may show large numbers of them and vice versa.

The liver of affected birds may show whitish or grayish spots evenly scattered or clustered in such a way as to suggest the presence of blackhead. Now and then typical blackhead lesions have been found in chicks four to eight weeks of age. In no instance have coccidia been found in livers showing either the grayish spots or the more or less typical blackhead lesions. Areas of perivascular infiltration with undifferentiated mesenchymal cells were of common occurrence in the birds studied. These areas might be thought to be due to secondary infection but very few positive bacterial cultures were obtained from livers showing such lesions. The livers showing blackhead lesions revealed a protozoan parasite (figure 12), now being studied by Doctor W. L. Chandler, parasitologist and protozoölogist at this Station.

A glistening rather uniformly brownish color of the heart was usually observed and the heart muscle was generally flabby. An increase in pericardial fluid was not uncommon. On microscopic examination there seemed to be more or less fatty degen-

eration in the muscle fibres and the musculature seemed loose in many cases.

Enlargement of nerves was observed quite often. Undifferentiated mesenchymal cell infiltration was found in the brain, spinal cord and peripheral nerves, some times even if no thickening of these nerves was noticeable.

Whether there is any connection between the changes noted in the heart and nervous tissues and coccidiosis infection is difficult to say.

## Summary

Coccidiosis in young chicks usually has its seat in the ceca and lower portion of the small intestines. The gross pathological changes are: Various degrees of hemorrhage and formation of more or less blood-stained, cheesy material in the ceca. Microscopically one sees: destruction of epithelium of the villi and gland tubules and sloughing of the mucous membrane. The cecal contents consist of tissue débris, necrotic material, blood cells and coccidia.

In birds over eight weeks of age one usually finds that the ceca are very seldom visibly affected. Duodenal infection is most common and the other parts of the small intestines may be involved at times. The gross pathological changes are: Thickening of the intestinal canal in the early stages and shrinking after sloughing of mucous membrane has taken place. Colonies of coccidia may appear as grayish spots or streaks in the mucous membrane, some times visible through the serous coat. There may be hemorrhages, dirty mucous exudate, ulcers and diphtheritic membrane. Microscopically we find that some coccidia seem to do little damage, infecting only single cells here and there. Others infect extensive areas of epithelium causing desquamation, some infect glandular epithelium as well as villous epithelium causing desquamation of both, and again we have those which cause "subepithelial"* infection and produce sloughing of the mucous membranes. It appears from this study that there are several species of coccidia, the *Eimeria tenella* and the *E. acervulina* having been quite definitely recognized.

Areas of infiltration with undifferentiated mesenchymal cells occurred frequently in the liver of infected chickens and always around the foci of infection found in the stroma mucosae. Many such areas were found showing no coccidia but it can not be

*As observed by Tyzzer, coccidia in the subepithelial tissues are evidently contained within enlarged epithelial cells.

stated with any degree of assurance that they always represent lesions of a more or less recent coccidiosis infection.

Leg weakness and paralysis occur more frequently in connection with duodenal than cecal coccidiosis.

Coccidia morphologically indistinguishable from some of those found in chickens were found in pigeons showing leg weakness. Coccidia were not found in the livers of infected chickens.

No evidence was found to show that sparrows carry the Eimeria.

## Acknowledgment

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## Descriptions of Figures

Figure 1. Very extensive "subepithelial" infection with *Eimeria tenella* of villi in the small intestines of an adult bird. Trophozoites, schizonts, gametocytes and oöcysts were found in these areas.

Figures 2 and 3. Deep-seated foci of coccidial infection. The bird from which these sections were obtained showed no coccidia in the intestinal contents. The intestines were shrunken, empty, and showed macroscopic evidence of sloughing of the mucous membrane. Note injury to the glands and muscularis mucosae and the extensive cell infiltration of the interglandular tissues and muscularis mucosae as well as the adjacent muscular coat.

Figure 4. Marked epithelial infection, with desquamation of the epithelium over the tips of the villi. Note the large oöcyst located just below the epithelium.

Figure 5. Moderate infection with *Eimeria tenella*, showing coccidia below the nuclei of the epithelial cells and just below the epithelium. Note the ragged appearance of the epithelium over the tip of the villus, the separation of the epithelium from the subepithelial tissues, and the cellular infiltration in the core of the villus.

Figures 6 and 7. Schizonts in interglandular tissue. Note the extensive cellular infiltration and the disappearance of the gland tubules, also the large number of schizonts in figure 7. *Eimeria tenella.*

Figure 8. Schizonts in interglandular tissues of cecum of chick. Infection of gland epithelium. *Eimeria tenella.*

Figure 9. (a) Tissue débris and oöcysts in lumen of a gland. Note the sloughing of the villous portion of the mucous membrane and cecal contents consisting of tissue débris, necrotic material and oöcysts. *Eimeria tenella.*

Figure 10. Massive infection of gland epithelium. Cellular infiltration in cecum of a chick.

Figure 11. Area of undifferentiated mesenchymal cell infiltration as commonly seen in livers of chickens.

Figure 12. Section from the liver of a chick showing blackhead lesions. Causative organism.

## Discussion

Dr. Oskar Seifried: May I ask if you found any lesions in the liver?

Dr. Stafseth: Only the blackhead type. I have never yet found coccidia in the liver.

Member: I would like to ask if you found any marked difference in the lesions in the small intestines and the cecum?

Dr. Stafseth: Yes, we did. I think Tyzzer was right when he described the different lesions as produced by different coccidia. One of the characteristic lesions we find consists of little grayish spots, sometimes visible on the serous surface, pin point to pinhead in size. Then, again, we have the typical transverse streaks, and in most instances in the early stages of coccidiosis thickening of the mucous membrane. Later there may be sloughing of epithelium with constriction. In a few instances we have the formation of a dirty grayish viscous mass, covering the mucous membrane and, occasionally, a diphtheritic membrane and ulcerations. However, in the cecum we find hemorrhages and ulcerations more pronounced than we do in the small intestines.

Dr. Geo. Kernohan: You spoke of small abscesses. Don't you find petechial hemorrhages there also?

Dr. Stafseth: Yes, we do.

Dr. W. T. Johnson: Just what is the basis for the determination as to the relation of coccidiosis to paralysis?

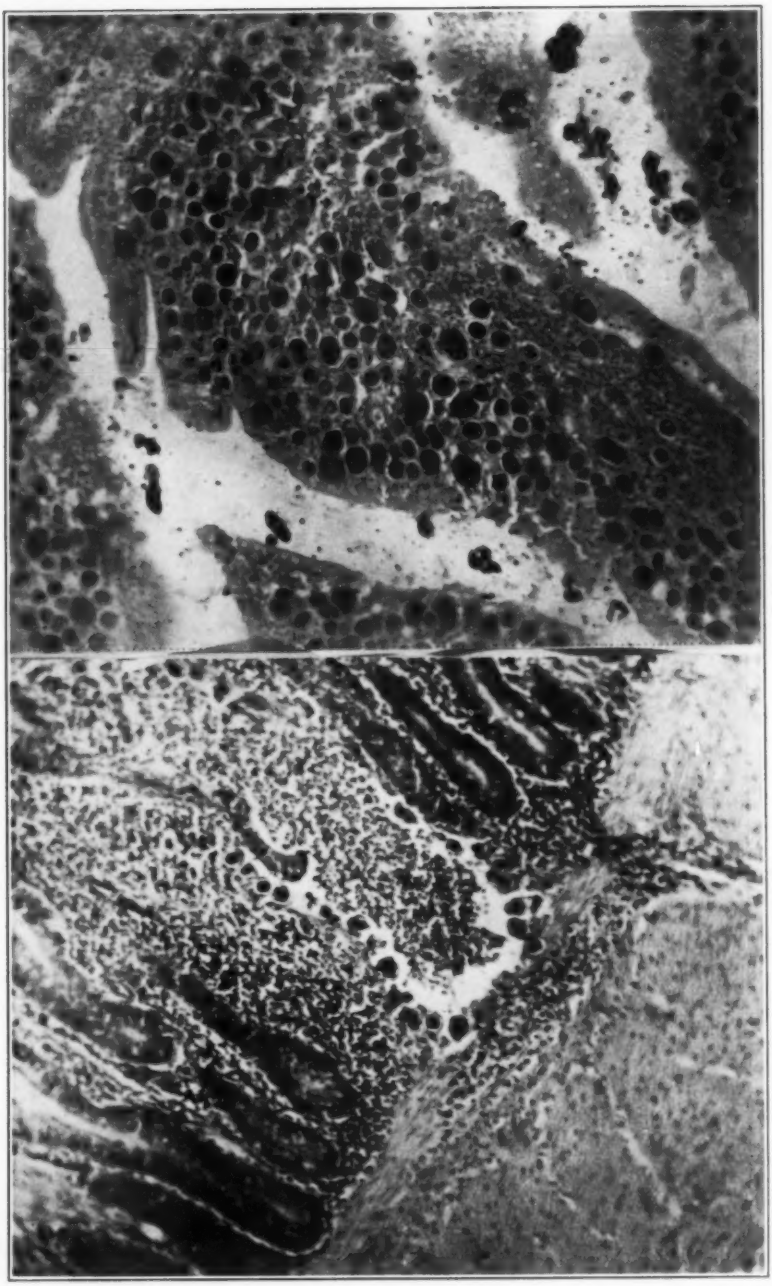
Dr. Stafseth: It is mostly statistical. I have taken three years of records and classified them and we find in the duodenal type, in chicks as young as four weeks, and in birds up to eighteen months of age, about 70 per cent of lameness, whereas, in chicks or adults that have just the cecal infection, only 3.4 per cent lameness, and I feel that while coccidia may not be the cause of it, they may open avenues of entrance to other microbes, which may exist mainly in the small intestines.

Dr. Johnson: Is that relation determined on the basis of various forms of coccidia?

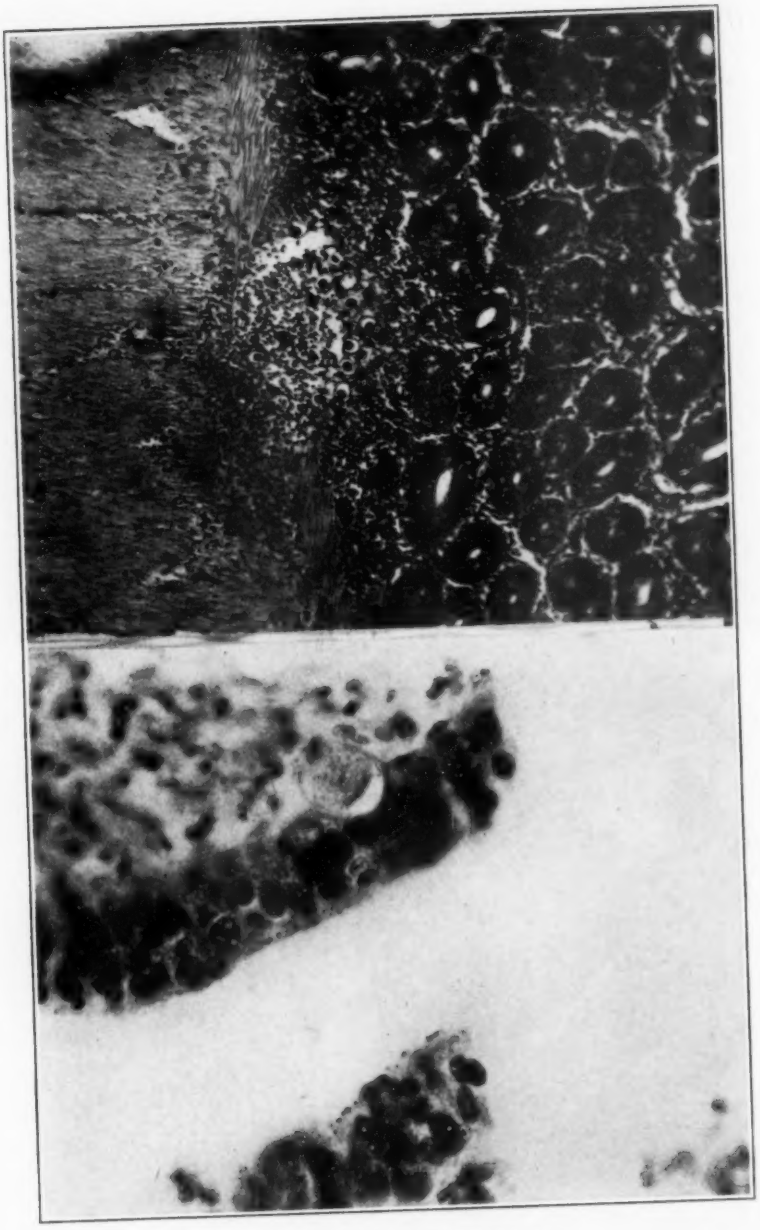
Dr. Stafseth: We find mostly what, in this paper, I have called the small type.

Dr. K. W. Niemann: Is it characteristic to find a different form of coccidium in the duodenum as compared with the cecum?

Dr. Stafseth: There is one type that is almost entirely located in the ceca —*Eimeria tenella;* another type, *Eimeria acervulina,* occurs mostly in the duodenal portion of the small intestines; *Eimeria maxima* is located mostly in the middle part and the so-called *Eimeria mitis* is found in scattered areas throughout the intestines.



FIGS. 1 (above) and 2 (below).



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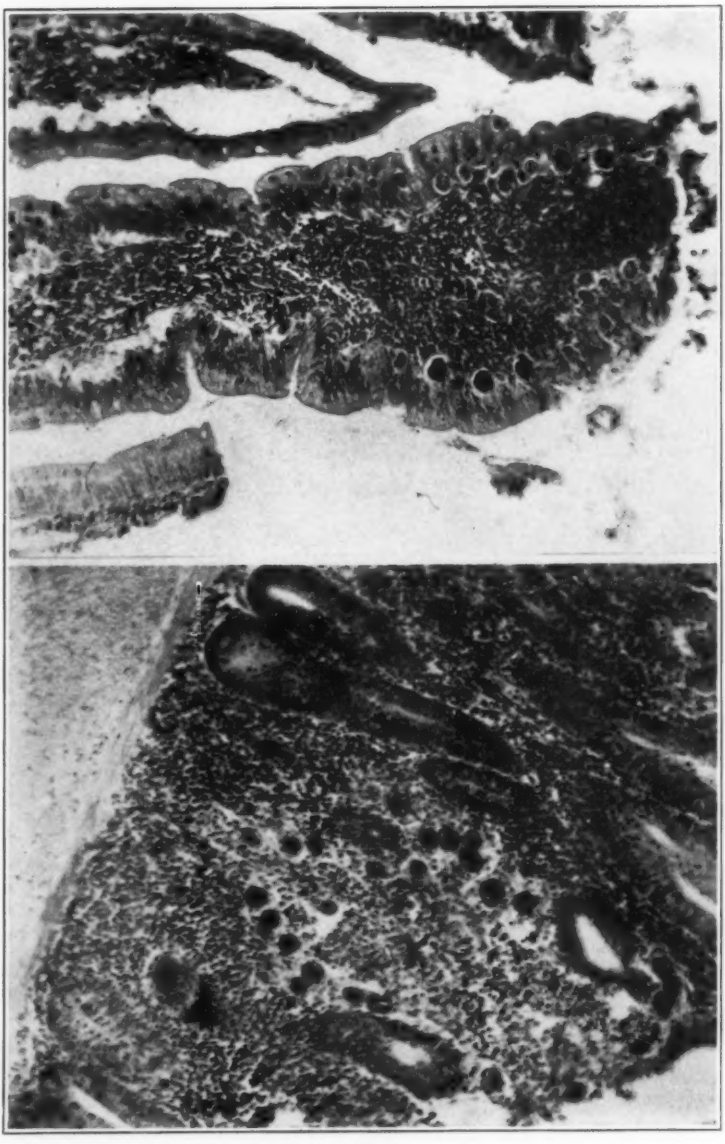

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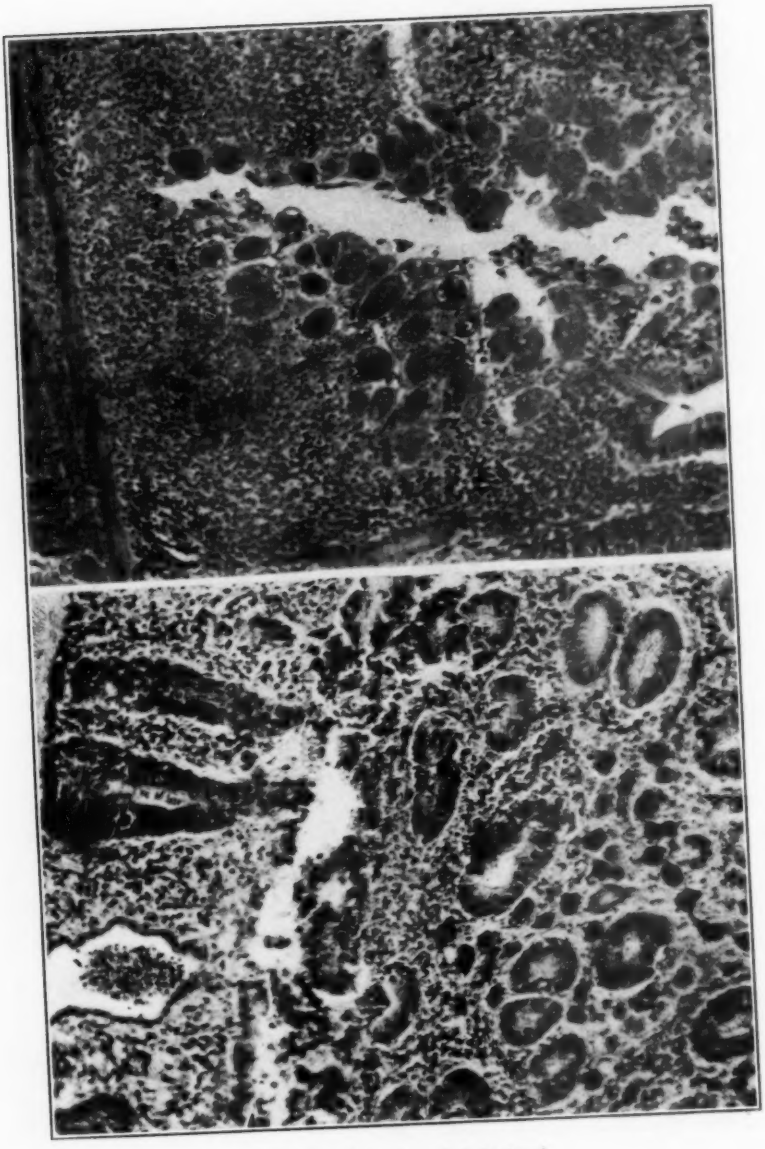

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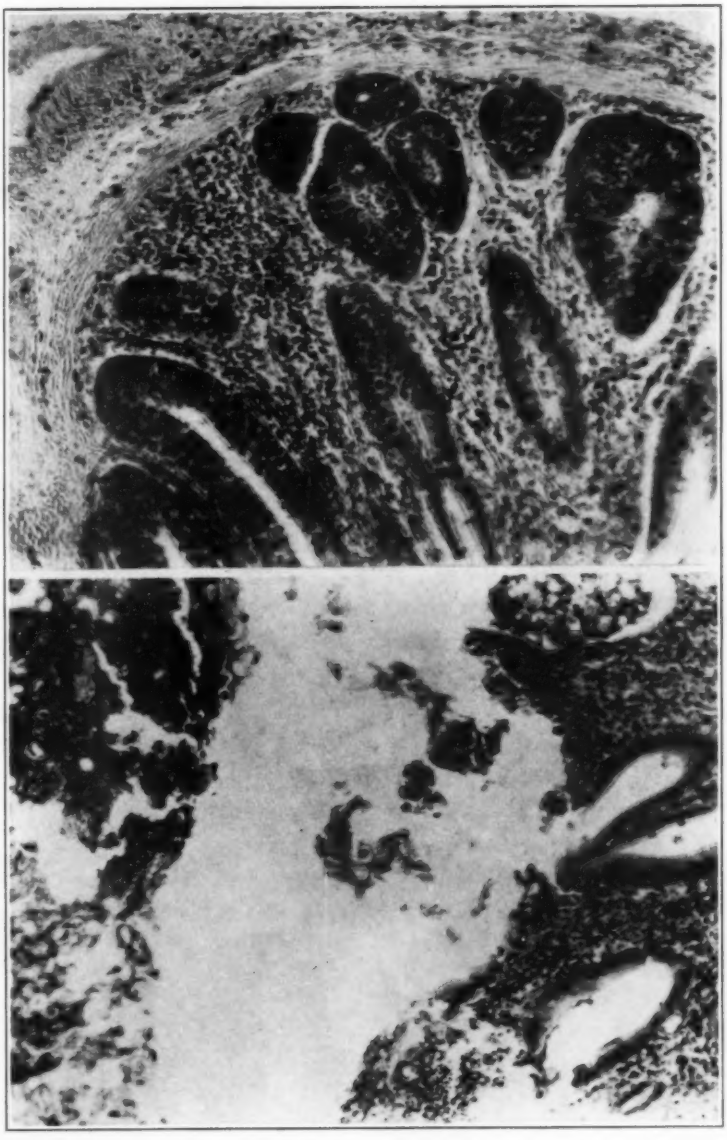

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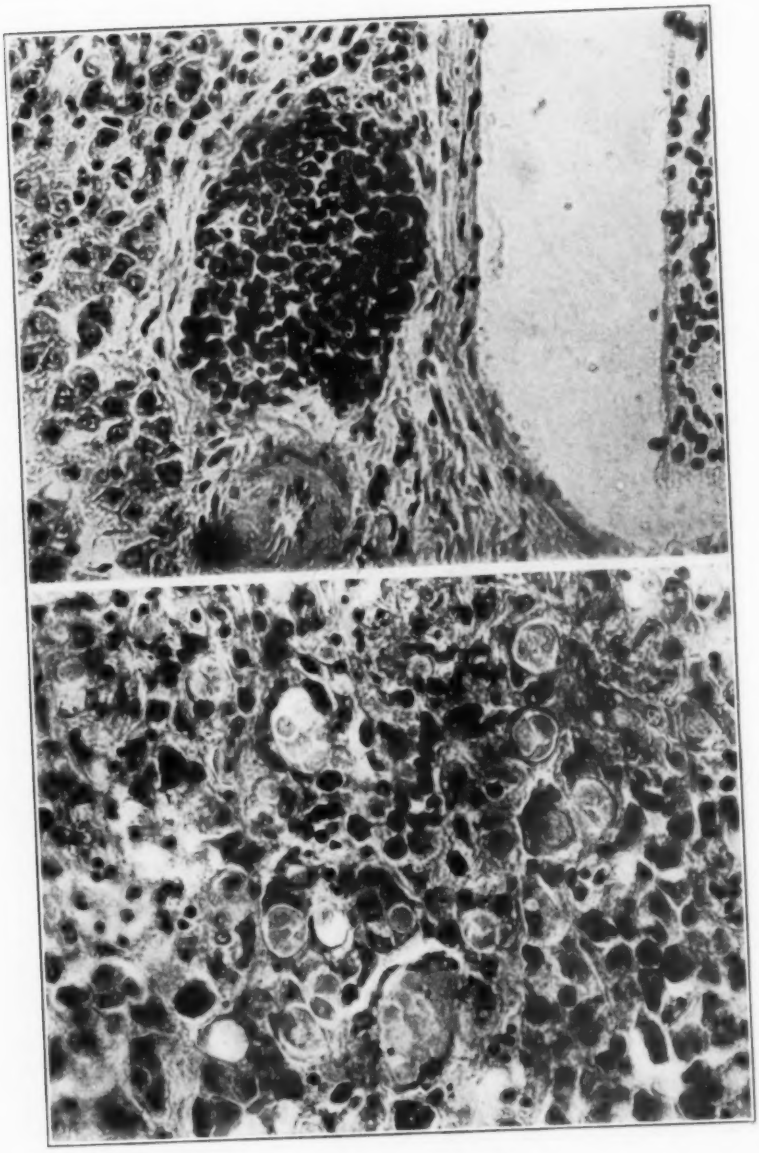

FIGS. 11 (above) and 12 (below).

# CLINICAL MANIFESTATIONS OF PATHOLOGICAL CONDITIONS OF THE GENITAL ORGANS OF SMALL ANIMALS*

*By* JAMES B. JONES, *West Hollywood, Calif.*

"Clinical manifestations of pathological conditions of the genital organs of small animals."

That's quite a title! It sounds like the beginning of a grand exposé of some of the hidden secrets of medicine or something left over from a couple of centuries ago, when everything about medicine was covered up with a cloak of black mystery—and a lot of long words. Modern medicine has no secrets—that is, practically none.

In reality, I take this title simply to mean diseased genital organs as we see them in everyday practice, such as a dog, for instance, that is a nymphomaniac, a cat that can not breed, a puppy attempting intimacy with a child, or conditions like these. I will not try to teach a thing new, but just try to bring out facts we all know.

Everyone realizes that this is an age of high-pressure efficiency. Man is exacting the maximum results from everything he manufactures. He demands that everything shall be bigger and better. Cars must run faster, skyscrapers must be taller, airplanes must climb higher. Invention, labor, materials, all are being pushed to their utmost capacity. Let's see what effect this trend of today is having on the animal world.

Half a century ago, on the cattle range, live stock were turned out at large. Cattle were left to their own resources. Instinctively the animals fed and watered themselves and the hand of man played only a small part in regulating their lives. All in all, they were healthy and sound.

Then we decided to breed for different types. We bred the horns off. We bred for this and we bred for that and from these highly bred cattle we expected a great deal—the maximum amount of milk, the maximum yield of beef and the maximum number of calves. Too much efficiency crept in. We bred for certain ideals and worked for certain ends—at the expense of health. We overcrowded them by asking too much. We

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sacrificed health for the sake of efficiency. We weakened the strain.

Today beef flesh is subject to diseases never heard of years ago. If such diseases were existent, cattle had the resistance to withstand them. The same decline has been brought about in dogs. Not so many years ago, dogs were working dogs or hunting dogs or watch dogs or just plain dogs, but they were not coddled or pampered or inbred. For the most part, people owned dogs and kept dogs for the work they could do.

If a dog was hungry, he went out and killed a rabbit or caught a bird. He might eat twice today and not at all tomorrow. If he happened to be around the house at meal time, he got some heavy coarse food which his rough mode of living enabled him to assimilate. Dogs were outdoors in rain and snow, and battled against the elements without dying of exposure. A female, ready to whelp, would crawl under the house and, without assistance, would turn out a litter of healthy puppies—puppies too rugged for the most part, ever to know the meaning of distemper.

## The Effects of Intensive Breeding

Even a quarter of a century ago, our dog population was healthier than it is today. We have taken dogs out of their natural mode of living and lavished them with care and—what is more important—we have begun intensive breeding. The dog that is just plain canine is encouraged no longer. We are commercializing this intensive breeding. Dogs are bred for types, bred to develop new types—inbred and cross-bred—all with the idea of turning out more dogs and more expensive dogs, with the strength of the strain a neglected consideration.

We are asking dogs to give more puppies and more puppies. We call on the female to produce one litter after another. We expect the male to breed day in and day out. By overproduction and by breeding for maximum efficiency in type, we are destroying resistance and health. We are guilty of starting race suicide in dogs. Just as with cattle, our intensive breeding of dogs has produced a weaker race, as evidenced by the number of diseases prevalent among them today. This, in a way, has accounted for the recent and rapid growth of the veterinary profession.

When the general body is weakened, is it not plausible that the genital system becomes impaired in direct proportion to the rest of the body? Assuming this is so, we have the explanation for the great number of pathological conditions of the genital organs

of the dogs of today—their congenital malformations and general predisposition to weakness and disease. There are, of course, any number of such conditions. Here we will deal with only a few which I believe to be interesting problems.

Many Bostons and Pekes, we find, are born with a congenital malformation of the prepuce. Some are without a prepuce entirely, others have simply a groove in the belly or an incomplete prepuce and the penis is exposed. Although this error in development seems more prevalent among Pekes and Bostons, a "wire" was once brought to us with an abnormally large preputial orifice. Every time he sat on his haunches, his penis protruded an inch. While standing up, it was quite normal.

We operated to correct the condition and closed the wound. Two days later the dog had licked the stitches out. In a matter of hours he had the orifice twice the size it was when brought to us. This was decidedly difficult to explain to the owner. We tried once more. We completely closed the existing orifice and made a new or false opening on the dorsal side. We kept the animal under a slight sedative for four days, during which time he did not lick the wound and scarcely even touched the area. In about ten days or two weeks we discharged him with what seemed to be a normal prepuce.

### Congenital Atresia of the Vagina

In the female an interesting type of congenital defect is atresia of the vagina. A female Collie, eighteen months old, was examined. The first time the Collie came in season, she was not bred. The second time, the owner tried to breed her to an excellent male, who did his best but could not complete the task. It seems that the male could insert his penis only part way and then would stop in pain.

After repeated attempts to breed her, the owner brought the female to the hospital. At first we were not definitely sure whether it was an impervious hymen or a case of atresia. The vagina inside was heavy and thick and had a dense, coarse feel. We could not clearly distinguish the cervix. From the vulva to the occlusion was a very short distance—a couple of inches, instead of three and a half or four, as would have been normal for a female of her size. We dilated with a speculum and introduced a light and definitely decided there was an atresia. We gave an unfavorable prognosis and the owner, to whom the

Collie was an investment for breeding, and not a pet, told us to put her to sleep.

We did so, held an autopsy and found congenital atresia—a complete occlusion of the wall of the vagina. The uterus itself showed all the characteristics of a pyometra, but further investigation proved that its contents was menstrual fluid, which was sterile. Although this Collie had been in season and evinced all the other signs, her history proved she had never passed any menstrual fluid. Her vulva had been swollen, however, and she had given every other manifestation during estrum except discharging.

The flow, never discharged from the uterus and still imprisoned there, was thick, viscid and bloody, with almost the consistency of honey. I have kept a specimen of this because I consider the case quite a rare example of congenital atresia. I must add that even if the owner had wished the dog to live, we could never have made it possible for her to become a breeder. We could only have suggested spaying.

### Cystic Ovaries and Nymphomania

A much more common condition affecting female animals is cystic ovaries, which causes a state of being chronically in season, known as nymphomania. Improper breeding, with its resultant weakness, often creates a predisposition toward this condition.

Cats with cystic ovaries, constantly in heat, cause untold annoyance to their owners and everyone else within earshot. One of our best clients was asked to move out of a fashionable apartment house on account of her Siamese cat which kept the neighbors awake all night with her serenades. The animal was afflicted with cystic ovaries, a typical nymphomaniac.

In the past few months, nymphomania in a certain female dog has created quite a sensation in this locality. It cost a male dog the sight of one eye and the State of California an expensive prosecution. In court the matter went on the docket as: The Case of the People of California vs. Chester Johnson.

Chester Johnson owned a female Boston with cystic ovaries. Being a nymphomaniac and constantly in season, she attracted a gang of male dogs to her owner's premises, much to the detriment of his goldfish, his flower-bed and tender young plants coming up in the garden. So Mr. Johnson got decidedly tired of that kind of fooling around and, with the little female, induced

three of the dogs into the garage, and—according to the evidence—gave them a good sound whipping. After that he poured household ammonia in the faces of two of the dogs.

One of these animals, a bulldog belonging to a Mr. Wilson, was treated in our hospital. The mucous membrane of his mouth sloughed off and both eyes were decidedly burned, the left one to such an extent that he lost the sight of it.

At the end of the three-day trial, Mr. Johnson was found guilty of inhumane treatment of animals and given a sentence of six months in jail and a fine of a thousand dollars. The sentence, nevertheless, was suspended, and the man put on probation. That is the only case I know of where nymphomania has had such far-reaching effects.

Cats and dogs with nymphomania are frequently sterile. I have seen female animals with cystic ovaries bred and rebred, yet never impregnated. Ninety per cent of the time the stimulus to the ovary is only cystic and the ovum is absent. Since an animal with cystic ovaries is apt to be no good as a breeder, there is really no disadvantage in spaying, which is the only cure or relief from nymphomania.

### Tumors of the Vagina

Other pathological conditions sometimes present symptoms almost identical with nymphomania. I refer to a ten-year-old Boston, supposed to be chronically in heat. She had a constant bloody discharge, thin, but very profuse. The supposition was that she was a nymphomaniac, although at first I even thought she was about to deliver puppies, because of an enlargement of the vulva and a continual straining that looked like labor pains. She had not been bred, however.

Examination revealed that the animal had a tumor of the vagina, getting gradually larger and larger. We discovered that the bloody discharge was caused by metritis. The tumor, an abnormal condition, which she was trying to relieve, had set up a reflex which looked like labor pains. This, together with the bloody discharge from metritis, produced the symptoms.

There was no choice of treatments. An operation to remove the tumor seemed the only thing to do. Although we were afraid that at ten years of age and in her depleted condition, she could not stand the operation, we took the chance and we were right!

Tumors found on the ovary are another cause of disturbances of the menstrual flow. I saw the case of an eight-year-old dog

which came in season every month or two. The cause was a thirteen-ounce tumor on the left ovary. On removal of this, the condition was corrected.

A wire-haired female was once presented for examination. The first time she came in season she had been bred and had delivered a litter of puppies normally. The second time she came in season her owner attempted to have her bred, but the male could not introduce the penis. The owner then took the female to a veterinarian who removed a tumor from the vagina.

The third time she came in season, the owner again attempted to have her bred, but with no success. Even though the tumor had been removed, the male still could not insert the penis. We examined and found a stricture of the vagina, due to scar tissue left by the removal of the tumor six months before. We operated to break down the scar tissue, and ordered the dog bred the next day—rather a heroic measure to prescribe—but we wanted it done before a local reaction had taken place. So the next morninb she was bred according to Hoyle. Sixty-three days later she whelped her puppies normally.

In addition to such cases of tumor of the vagina, there are, of course, numerous other conditions affecting the vagina, but these, as well as many affecting the uterus, I am omitting. They will, no doubt, be treated most capably in a paper which is to be given tomorrow.*

In the female dog, tumors seem to be the most prevalent in the breast. Surgical interference is the only means of relief. Since tumors are usually induced by irritation, we can assume that the nursing of puppies is a cause. Nursing one or two litters has, in the cases of some females, seemed to produce the condition, although often the tumors did not show for a year or so after nursing. In many cases these tumors are more or less a characteristic of age. In most mammary tumors of aged females, the veins are varicosed. I believe that the nursing of puppies and senility are causative factors of tumors of the breast, with varicose veins as a possible contributory factor.

In the male we find that tumors of the scrotum are by no means infrequent. They may or may not affect the testicle. In some cases the scrotal sac alone is involved, while in other cases the gland or spermatic cord may be affected.

An interesting case was a nine-year-old Boston with tumor of

*"Canine Obstetrics," by Dr. Horace A. Gould, published in the JOURNAL, May, 1931, pp. 634-650.

the scrotum, involving only the scrotal sac. We removed the tumor without invading the tunica vaginalis. Some time later we noticed the beginning of another tumor higher up, near the neck of the scrotum. As this tended to involve the spermatic cord, we recommended castration, with removal of all the diseased tissue.

Tumors attack the penis as well as the scrotum, often interfering with breeding and sometimes with urination. Occasionally the penis protrudes and can not be replaced, giving the appearance of paraphimosis. Among conditions affecting the genital organs of male dogs, paraphimosis has an important place. It is sometimes a serious and painful condition.

I remember the case of a little Peke, a chronic masturbator. Five times he has been brought to me with the penis in a state of erection, protruding and swollen too large to return to the sheath. Once the owner had to bring him in three times in one day for the same condition.

Another chronic masturbator, a seven-year-old Cocker, showed symptoms of pain and discomfort in the abdomen. He walked and acted as though he had a severe "tummy-ache." We filled him with barium and shot x-ray after x-ray, each showing a normal intestinal tract. We still treated him, however, for intestinal pain.

### Masturbation and Paraphimosis

One day I caught him licking himself in the act of masturbation. The penis was in a state of erection and he was going through moans and all other signs of orgasm. We changed our diagnosis to chronic masturbation, put him on sedatives and kept him away from females for days. We finally had to resort to castration.

A Boston which was addicted to masturbation and suffered subsequently from paraphimosis, was once brought to our hospital after the penis and bulbus glandis had protruded for three days. The poor animal was in terrific pain.

We worked with him for hours with alternating hot and cold packs trying to stimulate the circulation. We tried to replace the organ and sew the opening shut. We packed the penis for six hours with sugar and with the hope of drawing out the serum and blood. Next morning we scarified the region with multiple punctures. This plan did not work, but did allow infection to attack the penis where previous self-mutilation had paved the

way. The organ became gangrenous. Infection caused a sloughing of the whole penis down to the bone. For chronic paraphimosis castration is usually the only cure. It is only in diseased conditions that we like to recommend the castration of a male dog, whereas in the male cat we do recommend castration unless the animal is to be used for breeding purposes.

Spaying of the female, of course, is a much more frequent operation. An interesting case is a Poodle that I once spayed, quite successfully, I thought. She surprises me now by still attracting male dogs at given periods. She was spayed when approximately two months old. I personally did the operation, and I am quite positive—as positive as I can be—that I removed the uterus and the ovaries in their entirety.

Nevertheless, when about a year old, this Poodle was discovered in the company of five male dogs, and she seemed rather flattered by their attention. After a week there were no more dogs hanging around. Six months later, another horde invaded the neighborhood, all seeking to be in the company of this little Poodle. To my knowledge there were no attempts at copulation. She is now two and a half years old, and approximately every six months since the first visitation, the same phenomenon has taken place. If everything goes true to form, a repetition of it is to be expected soon. Although periodically attracting these males, the poodle has never discharged nor evidenced any signs of estrum.

### Ovaries Generate Sex Hormones

It is a settled fact that if we remove the ovaries, we remove the generating plant of sex hormones. This dog had her organs removed before they ever became active. The question in my mind is whether or not her glands are brought into play in the absence of the ovaries or whether I actually failed to remove all of the ovarian tissue. I have heard of several cases of the same thing which so far have not been explained.

Another phenomenon occurs which is most interesting and at the same time difficult to explain. An example of this is Topsy, an Airedale—or rather she would be an Airedale if she were not so mixed. On February 23 of this year, Topsy came to our hospital to board while she was in season. In due time she was discharged and sent home.

Topsy positively had not been bred. Yet, about forty days after she first came in season, she showed an enlargement of the

abdomen and a stimulation of the mammary glands. About seventy-three days from the date when she first came in season, she began to show signs of nervousness and anxiety. Her breasts had formed and milk had formed within them.

On May 8, exactly seventy-five days after she came in season, Topsy went through all the disturbances of whelping. Not a detail was missing—except the puppies. This condition passed away in a day or so, but her breasts stayed completely full of milk and later became caked. Three or four times a day we had to milk Topsy like a little cow and treat the breasts. Oftentimes the breasts swell a bit about sixty days after the heat period, but in Topsy's case the swelling was decidedly abnormal. And a bountiful supply of milk was present. It is apparent that Topsy underwent a pseudo- or phantom pregnancy. One very important way in which Topsy's condition tallied with a dog that had whelped, is that Topsy came in season again, not six six months from the date when she had previously been in season (which would have made it August 1) but exactly four months after her imaginary delivery (on August 16). If she actually had been bred on the twelfth day of her estrum (March 7), and had carried her puppies sixty-three days, her calendar would have been marked just as it is now.

### Vaginitis and Metritis

The sex life of a dog, particularly a female, is not all milk and honey. She takes her risks, the same as anybody else. One of the risks of copulation is the possibility of acquiring vaginitis or metritis. This condition may be caused by infection during difficult labor or by improper assistance at that time, but it may also spring from copulation with a male having a catarrhal discharge of the sheath. And in ninety-nine per cent of all male dogs this condition exists to a greater or lesser degree.

Catarrh of the sheath, or preputial catarrh, although called gonorrhea by the layman, is not a true venereal disease. However, although exceedingly prevalent, the condition is not normal. It is sometimes caused by an irritation from the passage of uric acid, and the constant licking which this irritation brings about.

Recently there was brought to my attention what I considered a rare case of rape. The owners of a wire-haired female called up and said their dog was in the act of copulation. They stated that she had no reason to be because it was two months ahead of

the time she was supposed to come in season. Yet they had witnessed her union with a male.

They brought her to the hospital immediately. We examined the vulva and the vagina. About an inch inside the vagina we found a foxtail, which we removed. About an inch further inside the vagina we discovered a rent or tear in the wall It is obvious that the foxtail could not have caused this rupture. Therefore, it must have been caused by the forced entrance of the penis of the male. It seemed improbable that the male dog, which was only a little bigger than the female, could have ruptured her vagina in this way if it had been a normal breeding. My theory is that the foxtail in the vagina, acting as a local irritant, produced much the same effect as cantharides, which is an aphrodisiac, and that it set up enough irritation to arouse sexual excitement, even though the female was not physiologically ready to breed. The introduction of the male's penis soothed the irritation and she allowed this intimacy. In so doing, she was ruptured because her organs were not ready for the mating. There was not the natural elasticity which would have existed, had she been in season.

### Effect of Castration and Spaying

A problem concerning the genital organs which crops up in almost daily practice is the effect of castration and spaying on the general physical condition of dogs.

Whether or not the animal will become fat and lazy after the operation is an excellent topic for debate and is probably a matter of opinion. Personally, for every female anyone can show me that got fat after spaying, I can show him one hundred that did not. That is, providing the animal is given proper exercise and proper diet. On the other hand, for every male you can show me that did not get phlegmatic and fat after castration, I can produce one hundred that did.

I cannot help but wonder if the reason is psychic. Can it be that since the female's sexual attitude is mostly acquiescence, the loss of her sex organs does not necessarily deprive her of her main interest in life, and she still enjoys running aroung enough to keep her weight down? The male, however, being naturally the aggressor in the sex act, may, when he is robbed of his sex organs, lose what is the inspiration for all his energy. May that not account for what he does? He lies down and gets fat and sleeps his life away like the eunuch that he is.

Another problem which may involve the psychological angle is the practice of masturbation by the dog. We know that most baby puppies, three, four, or five months of age, go through some of the motions of the sex act without a partner. This is not a complete masturbation as we see it in older dogs, which are chronic perverts. The puppy sometimes attempts to mount an inanimate object, or the arm or foot of a human, or sometimes just assumes the position and goes through the motions. It is not a complete masturbation, in that it is seldom carried to the point or orgasm or satisfaction.

### Pschycho-Analyzing the Growing Puppy

I imagine such demonstrations are psychic, caused by a dawning consciousness of sex. If we chose to step into an ultramodern trend and psycho-analyze the growing puppy, we might advance certain tentative theories about the source of this manifestation. In the first place we notice that a puppy does not start this practice until after he has stopped nursing. Now one school of psychiatry informs us that a human baby, nursing at a mother's breast, enjoys a definite sexual satisfaction from the act. Is it not possible then, that the puppy, kneading at his mother's udder while nursing, derives a similar sex stimulation, and that when he is weaned and this pleasure is denied him, he naturally craves some form of sexual expression until he becomes mature enough to focus his interest on copulation?

We know, also, that a puppy will almost invariably single out a woman's foot or arm to rub against rather than a man's. Might not this fact, then, back us in the conjecture that the sensation of nursing leaves a trace of the so-called "mother fixation" in the puppy's sex phantoms, which still persists after he turns to masturbation and which leads him to seek feminine proximity?

Be that as it may, the puppy that discards masturbation when he loses his puppy teeth presents no problem. The difficult ones are the renegades who carry the practice with them into maturity.

Medicine, so far, has no valuable explanation to offer of perversion in adult humans, nor can we really explain this practice in dogs. But we do know that the morals of animals are usually high because animals normally follow the dictates of nature.

Is it not possible, then, that by weakening our dogs physically, we have weakened them morally? Is it not reasonable to believe that if we gave them back their normal mode of living, we would develop a stronger strain morally, as well as physically, and

thereby do away with many of the pathological conditions of the genital organs?

DISCUSSION

DR. G. W. MCNUTT: I believe I can answer a question that seems to be pressing Dr. Jones and that is in reference to the bitch which comes in heat after spaying. Of course, there is always a possibility of leaving a portion of the ovary, but that operation can be completely performed and we still have a bitch coming in heat after she has been spayed. This is the result of ovarian tissue misplaced in development. It is a congenital condition. These small particles of ovarian tissue are found principally in the broad ligament, or in the vicinity of the ovary. Sometimes they are not removed when you take the ovary out. That is not common in dogs, but it does occur.

I recall one case in particular, of a bitch spayed by an experienced operator, and she was brought back to the hospital a year later. The owner said that the bitch was coming in heat and that he was going to give her to the hospital. The dog was autopsied and I found on the cut horn of the uterus two small brownish-yellow bodies; on sectioning these they showed typical ovarian tissue and corpora lutea, showing that ovulation had taken place in these new developments.

I believe that the majority, probably, of these cases which come in heat after they have been spayed are due to misplaced ovarian tissues. That is particularly true when the operation is performed by an experienced surgeon, and he is quite sure he has removed both ovaries. In spaying mice for the sex hormone test, Parkes states that 60 per cent will, in time, come in heat again, due to misplaced ovarian tissue. Frank states that he has reduced this percentage to less than 1 in 200 by removing as much as possible of the surrounding peritoneum.

There is one other point which might be of interest with reference to dogs getting fat after castration. Of course, in a masturbating dog this wouldn't be possible, at least it is questionable whether it would, but in a dog from which you are removing the testicles, because of tumors, if you should find a portion of these testicles, even a small part, which is normal, it could readily be placed back in the animal. If the whole scrotum is removed it could be placed in the muscle where the blood supply is rich and it will serve the purpose of sex hormone in that animal for a number of years.

Autotransplants are practically 100 per cent successful. Homeotransplants might be tried; they are successful in a majority of cases, but autotransplants of the sex tissue are practically 100 per cent successful. That is one thing that we might try, in case of tumors involving the scrotum and the sex organs of males.

DR. J. J. HOGARTY: I was very much interested in the paper read by Dr. Jones and I think it is a very important thing from the standpoint of practitioners.

I would like to relate a case, involving a cat. This cat was spayed, both ovaries removed, and about two months afterwards she was quite a nuisance, roaming around, yelling, and keeping everybody awake at night. She was brought to the hospital by the owner. I looked her over, suggested that she be given a little more time, and told the owner to bring her back later. The cat was brought back. I opened her up and she possessed a supernumerary ovary and uterus. The ovary was cystic and I found both stumps of the uterus where the ovaries had been removed. A cyst had developed. Both stumps were removed, with the extra uterus and ovary, the cat has entirely recovered and is now normal.

The doctor just mentioned about cells being found there. I would like to say that when spaying you might use the light and be sure there isn't an extra ovary and uterus there.

A bitch was spayed when five months old, a collie. She gave birth to one pup, nine months after the operation, with a prolapse of the uterus. She was dragging this around on the ground. I might say that the owner, a lady, threatened to sue us. Dr. Archibald was alive at that time and I described it to him. He told me to get the bitch back and operate on her, which I did, and

I received a good roasting from the lady. But the bitch recovered. I saw her when she was nine years old. She never had any further trouble. That was another case of supernumerary ovary.

I have those specimens and I am going to show the cat specimens to prove that both ovaries were removed.

DR. MCNUTT: The congenital tissue which we find placed in the broad ligament, or in that vicinity, during the life of the natural ovaries, and when they are present, is so small that we usually cannot see it. That would be left in there, possibly, because it is so small we wouldn't recognize it.

DR. HOGARTY: For the benefit of the younger men I might say that I remember at one clinic a bitch was presented that had been spayed and she was still nymphomaniac and was passed up to a surgeon to operate on her. He asked me if I would assist him. I told him there was only one thing to look for and that was a piece of ovary. We found that an ovary had been left in on one side. We removed that. But, to show you how you make a mistake, we didn't look on the other side; he might have left the ovary in and still the bitch would be a nuisance. So we cannot be too careful.

DR. J. C. MCGRATH: I never had a bitch come in heat after I thought I had spayed her, but I narrowly escaped it a short time ago. I hurriedly spayed a bitch and, as I laid down the second ovary, I noticed it wasn't just exactly uniform with the other. I went to lunch, commenced thinking about the matter, and on my return I looked again and I found the ovary that should have been taken out. The other tissue proved to be a small fibrous tumor. There was a case where one ovary could have very easily been left in and the bitch would have come in heat again. That is another thing that might be looked for.

## Minnesota Veterinarians to Meet at University Farm

Secretary Fitch announces that the summer meeting of the Minnesota State Veterinary Medical Society will be held as a joint meeting of the organization and a short course at University Farm, Saint Paul, July 2 and 3. The following prominent veterinarians will contribute to the program: Dr. Maurice C. Hall, president of the A. V. M. A.; Dr. D. H. Udall, of Cornell University; Dr. I. F. Huddleson, of Michigan State College; Dr. J. A. Campbell, of Toronto, Canada; Dr. R. L. Cochran, of Ames, Iowa; Dr. Herbert Lothe, of Waukesha, Wis.; Dr. A. H. Quin, of Des Moines, Iowa; and Dr. H. B. Treman, of Rockwell City, Iowa.

## Bureau Veterinarians Study Ethics

The National Association of Bureau of Animal Industry Veterinarians has decided to have a code of ethics for the guidance of the activities of the members of the organization. A committee has been appointed to draw up a code of ethics and a tentative draft appeared in *The Bureau Veterinarian* for March. The committee which has this matter in charge consists of Dr. E. M. Nighbert, Chairman; Dr. Frank Jelen and Dr. D. R. Gillies. The proposed code will be presented for adoption at the 1931 annual meeting.

# BRUCELLIASIS IN THE SWINE HERD OF THE UNIVERSITY OF CALIFORNIA*

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Brucelliasis in swine has existed in the University of California herd, in an enzoötic form, for many years, but without marked visible effects of the infection until recently, when abortions and reactors increased to an alarming extent. At least two human cases of undulant fever have been traced to contact with this herd. It is believed timely to report the findings and observations made on this herd, since the publications of Hayes and Traum,[1] and Hayes and Phipps.[2]

The swine herd of the University of California, at Davis, was started in 1908 and the raising of swine has gone on continuously since that time. In the year 1918, abortions appeared in the herd, and all animals reacting to the agglutination test were sold. In the year 1920, abortions appeared in the herd again; and work was carried on with this disease through the year, reports of which were published by Hayes and Traum in their article dealing with three outbreaks of swine abortion in California, in which an organism of the type of *Brucella abortus* (Bang) was the probable causative factor. The organism was readily isolated from the chorion and from the stomach contents of aborted pigs. Agglutination tests of the swine indicated that the sows giving strong reactions may give birth to apparently normal litters and that, furthermore, the blood titre is variable and tends soon to decline.

However, no further serious trouble was encountered with brucelliasis in this herd until January, 1929, when several of the sows aborted. Agglutination tests of the blood of all the animals in the herd were made, and most of the positive reactors were removed from the herd and slaughtered. Several of the reactors were not removed, because these animals were on an inbreeding experiment which has been going on continuously for the past ten years. Other positive animals on short-time feeding experiments also were allowed to remain in the herd until the completion of the projects.

*Presented at the sixty-seventh annual meeting of the American Veterinary Medical Association, Los Angeles, Calif., August 26-29, 1930.

Since January, 1929, frequent agglutination tests and other observations have been made, the results of which form the basis of this paper.

## Methods of Procedure

*Collection of blood samples:* The blood samples to be used for the agglutination tests are obtained by collecting blood from one of the veins on the outside of the ear. The ear is cleansed with a 70 per cent solution of alcohol, and an ear vein punctured with a sharp-pointed knife. Some of the animals are bled by cutting off a small piece of the tail. Precaution is used in cleaning and disinfecting the knives after each bleeding. The containers used in collecting the samples are small test-tubes about 4 x ½ in. The tubes are dry sterilized, carefully corked, and labeled.

From this point on, the procedure follows that described by the University of California Committee for the Study of Brucella Infections:

*Preparation of antigen:* Antigen used in all the tests is made from *Brucella abortus* U. C. culture 80. Washing and preserving of this antigen is as follows:

Wash off the growth with saline solution containing 0.5 per cent phenol, care being exercised not to scrape up particles of medium into the suspension. The lips of the culture tubes or bottles are carefully flamed and the concentrated antigen poured into the sterile containers. After storing the concentrated antigen for seven days at room temperature, away from any direct source of heat, tests for sterility upon agar medium are made. This concentrated antigen is then diluted with phenolized 0.5 per cent saline and standardized to Gates, 3.5—4.0, which corresponds to turbidity 2.5—3.0 McFarland scale, or a turbidity of 20,000 (Evans[3]).

### Agglutination Tests

*Sera:* Four tubes representing four dilutions are used in the test. Controls with known positive serum, known negative serum, and antigen alone are included in each set-up. The dilutions suggested are 1:25, 1:50, 1:100 and 1:200. Into the first tube of each series of four, place 2.4 cc of antigen. Into tubes 2, 3 and 4 of each series, place 1 cc of antigen. Into tube 1, place 0.1 cc of the serum to be tested. It should be free from blood cells. The contents of tube 1 should be rinsed back and forth by means of a pipette or rheometer syringe, to assure thorough mixing of the dilution. Transfer 1 cc of material from tube 1 to tube 2. Rinse thoroughly the contents of tube 2 and transfer 1 cc to tube 3. Proceed in a like manner with the contents of tube 3, and transfer 1 cc to tube 4. After mixing this material, discard 1 cc from tube 4. Use a fresh pipette for each serum, or rinse well to render clean. The dilutions are now 1:25, 1:50, 1:100 and 1:200, in tubes 1, 2, 3 and 4, respectively. Place the racks in the incubator.

*Incubation and reading of the tests:* Incubation should be at 37.5°C. overnight; then allow to stand at room temperature for one hour, and record results, after which the tubes in their racks are to stand *24 hours longer* at room temperature for a second reading. This holding process should be in a shady part of the room, away from any direct source of heat.

The interpretation of the tests is based upon the clearing of the supernatant fluid and distribution of sediment at the bottom of the tube. A complete positive agglutination is characterized by a water-clear super-

natant fluid and the arrangement of a flattened film at the bottom of the tube.

A completely negative agglutination is manifested by a turbid supernatant fluid and a central, compact, small, sedimented spot of organisms in the bottom of the tube.

A partial agglutination is a combination of the above with a turbidity less than negative and a smaller compact central mass with a zone of agglutinated bacteria around it.

At present, an agglutination higher than 1:50 is considered positive.

An agglutination of 1:25 but not higher than 1:50 is subject to retest. Retests should be made between two and four weeks from the previous bleeding.

The correlation of serological findings and the presence or absence of infection in cattle have been rather extensively studied. Such studies, however, in goats and in swine, have not been sufficiently large to guide us in interpretation. Accordingly, for the present, interpretations are based on the available data on the study of Brucella infections in bovines.

*Explanation of symbols:*

Decision column:

P, positive. This indicates that the animal is at present, or was at some previous time, infected.

N, negative. This indicates an animal probably not infected.

S, suspicious. This test is inconclusive, and the animal should be retested.

Tube readings column:

+, complete agglutination (indicated by the sign # when typewritten).

±, incomplete agglutination (indicated by the letter I when typewritten).

∓, trace of agglutination, less than ± (indicated by the letter t when typewritten).

−, no agglutination.

*Explanation of agglutination test record:* Each of the four symbols opposite the animal's number represents the degree of agglutination in that dilution of the blood serum of that animal. Reading from left to right, the dilutions are 1:25, 1:50, 1:100 and 1:200.

An animal whose blood serum causes complete (+) agglutination in the first two dilutions, together with any agglutination (∓ or higher) in the third or fourth, is considered positive under the system used at present at the University of California. An animal which gives no agglutination, or only an incomplete or trace of agglutination (± or ∓) in the first dilution, is considered negative. Those tests falling between negative and positive are considered as suspicious.

Following are typical readings and results:

| Dilutions | | | | Decisions |
|---|---|---|---|---|
| 1:25 | 1:50 | 1:100 | 1:200 | |
| + | + | + | + | P |
| + | + | ± | − | P |
| + | + | ∓ | − | P |
| + | + | − | − | S |
| + | ± | − | − | S |
| + | − | − | − | S |
| ± | ∓ | − | − | S |
| ± | − | − | − | N |
| ∓ | − | − | − | N |
| − | − | − | − | N |

### Guinea Pig Inoculation

Guinea pigs may be inoculated either subcutaneously, intraperitoneally, or intramuscularly. At the end of six weeks, the guinea pigs are chloroformed or gassed. At this time blood is collected for the agglutination test. Ordinarily, cultures from the spleen suffice, these to be made on cooked-blood agar or liver-infusion agar and grown under ordinary atmospheric condition and also under a condition of approximately 10 per cent $CO_2$ from a tank.

## Repeated Agglutination Tests on Swine Herd From January 18, 1929, to August, 1930

On January 18, 1929, the first agglutination test of the swine herd was completed, and all animals reacting to this test as positive or suspicious were sold for slaughter, except the animals on experiment, which were allowed to remain in the swine herd in a state of partial isolation. The same procedure was followed after each successive test up until March 10, 1930, when a tract of land about one mile distant from the hog barns was obtained for an isolation unit. Since March 10, 1930, all animals reacting as positive or suspicious to the agglutination test, except those on experiment, have been moved from the main swine herd and placed in isolation.

On July 23, 1930, another isolation unit was obtained for all experiment animals reacting as positive or suspicious to the agglutination test, in this way leaving the main hog unit for negative swine only.

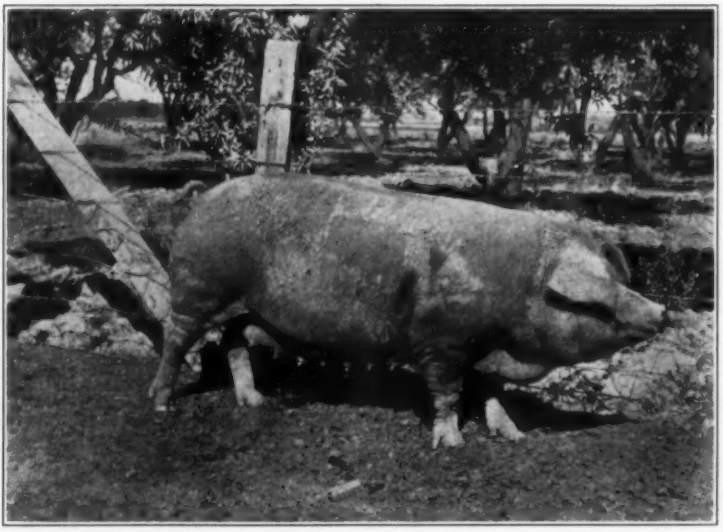

Fig. 1. Sow PC9F28, infected with brucelliasis. *Brucella abortus* (suis) culture 3 was recovered from the submaxillary and atlantal lymph-glands of this animal after autopsy.

TABLE I.—*Results of the agglutination tests in which reactors were removed by slaughter or isolation after each test*

| DATE | ANIMALS | NEGATIVE | | SUSPICIOUS | | POSITIVE | | REMARKS |
|---|---|---|---|---|---|---|---|---|
| | | No. | % | No. | % | No. | % | |
| 1-18-29 | Sows | 35 | 66.04 | 3 | 5.60 | 7 | 13.36 | Animals reacting P and S sold for slaughter |
| | Barrows | 2 | 3.80 | 0 | 0.00 | 0 | 0.00 | |
| | Boars | 3 | 5.60 | 0 | 0.00 | 3 | 5.60 | |
| | | 40 | 75.44 | 3 | 5.60 | 10 | 18.96 | |
| 6-10-29 | Sows | 42 | 77.80 | 2 | 3.71 | 6 | 11.11 | Animals reacting P and S sold for slaughter |
| | Barrows | 0 | 0.00 | 0 | 0.00 | 1 | 1.85 | |
| | Boars | 3 | 5.53 | 0 | 0.00 | 0 | 0.00 | |
| | | 45 | 83.33 | 2 | 3.71 | 7 | 12.96 | |
| 11-1-29 | Sows | 18 | 37.50 | 4 | 8.33 | 19 | 39.59 | Animals reacting P and S sold for slaughter |
| | Barrows | 0 | 0.00 | 1 | 2.08 | 0 | 0.00 | |
| | Boars | 6 | 12.50 | 0 | 0.00 | 0 | 0.00 | |
| | | 24 | 50.00 | 5 | 10.41 | 19 | 39.59 | |
| 1-14-30 | Sows | 18 | 54.55 | 3 | 9.09 | 7 | 21.21 | Animals reacting P and S sold for slaughter |
| | Barrows | 0 | 0.00 | 0 | 0.00 | 0 | 0.00 | |
| | Boars | 5 | 15.15 | 0 | 0.00 | 0 | 0.00 | |
| | | 23 | 69.70 | 3 | 9.09 | 7 | 21.21 | |
| 3-6-30 | Sows | 40 | 43.48 | 5 | 5.44 | 13 | 14.13 | Animals reacting P and S removed to isolation unit |
| | Barrows | 26 | 28.26 | 3 | 3.26 | 2 | 2.17 | |
| | Boars | 3 | 3.26 | 0 | 0.00 | 0 | 0.00 | |
| | | 69 | 75.00 | 8 | 8.70 | 15 | 16.30 | |
| 5-23-30 | Sows | 37 | 63.79 | 0 | 0.00 | 1 | 1.73 | Animals reacting P and S removed to isolation unit (other than experiment) |
| | Barrows | 16 | 27.59 | 1 | 1.73 | 0 | 0.00 | |
| | Boars | 2 | 3.43 | 0 | 0.00 | 1 | 1.73 | |
| | | 55 | 94.81 | 1 | 1.73 | 2 | 3.46 | |
| 7-3-30 | Sows | 56 | 50.00 | 1 | 0.89 | 1 | 0.89 | Animals reacting P and S removed to isolation unit (other than experiment) |
| | Barrows | 41 | 36.61 | 2 | 1.79 | 2 | 1.79 | |
| | Boars | 8 | 7.14 | 0 | 0.00 | 1 | 0.89 | |
| | | 105 | 93.75 | 3 | 2.68 | 4 | 3.57 | |
| 7-17-30 | Sows | 53 | 50.48 | 2 | 1.91 | 1 | 0.95 | Animals reacting P and S removed to isolation unit (other than experiment) |
| | Barrows | 40 | 38.08 | 1 | 0.95 | 2 | 1.91 | |
| | Boars | 6 | 5.72 | 0 | 0.00 | 0 | 0.00 | |
| | | 99 | 94.28 | 3 | 2.86 | 3 | 2.86 | |
| 7-31-30 | Sows | 44 | 43.56 | 7 | 6.93 | 3 | 2.97 | Animals reacting P and S removed to isolation unit |
| | Barrows | 32 | 31.68 | 4 | 3.97 | 3 | 2.97 | |
| | Boars | 7 | 6.93 | 0 | 0.00 | 1 | .99 | |
| | | 83 | 82.17 | 11 | 10.90 | 7 | 6.93 | |
| 8-14-30 | Sows | 45 | 54.22 | 0 | 0.00 | 0 | 0.00 | Animals reacting P and S removed to isolation unit |
| | Barrows | 32 | 38.55 | 0 | 0.00 | 0 | 0.00 | |
| | Boars | 6 | 7.23 | 0 | 0.00 | 0 | 0.00 | |
| | | 83 | 100.00 | 0 | 0.00 | 0 | 0.00 | |

A review of table I shows that even after a majority of the positive and suspicious animals were removed and slaughtered, and after the positive and suspicious experiment animals were held in partial isolation in the main hog unit, new reacting animals were found (with the exception of the last test made August 14, 1930), in each succeeding test.

### Results of Repeated Agglutination Tests on Reactors

Swine reacting as positive or suspicious to the agglutination test, other than the experiment animals, have been removed to the isolation unit and held under close observation. The attendants who take care of these animals do not come in contact

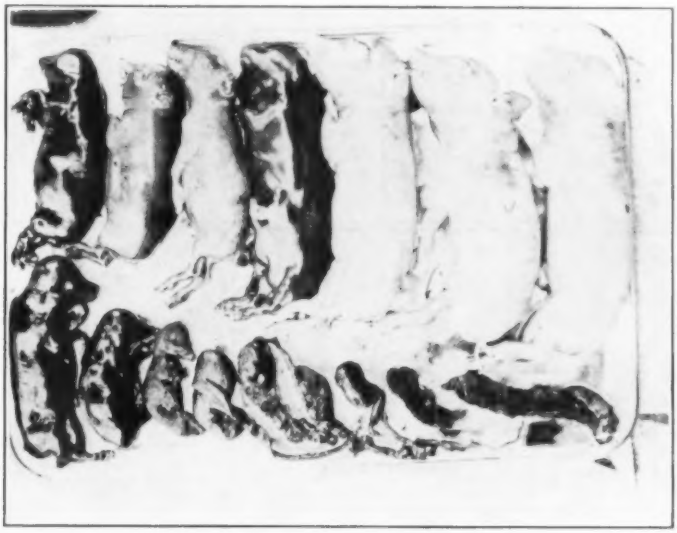

Fig. 2. Three apparently normal and 13 mummified fetuses, removed from uterus of sow DJ55F28 after autopsy. This sow reacted "suspicious" to the agglutination test.

with the negative herd. Repeated blood tests are taken of the animals at the same intervals as in the case of the main herd. The variations in blood titre and other observations are noted in table II.

A review of table II shows that the blood titre of these animals recedes at the time of farrowing and then has a tendency to rise to a level as high as before. Most of the sows have farrowed at the normal time. The percentage of pig crop and the number of live pigs born have been as large as in the case of an equal number of negative sows, if not larger. The percentage of positive

TABLE II—*Results of repeated agglutination tests on positive swine*

| IDENTIFICATION | DATE OF TEST | BLOOD TITRE | DATE OF BREEDING | DATE OF FARROWING | LIVE PIGS | DEAD PIGS | CULTURES | GUINEA PIG BLOOD TITRE |
|---|---|---|---|---|---|---|---|---|
| DJ2F29 Sow | 3-10-30 | + + + + | Not bred | | | | | |
| | 5-4-30 | + + ∓ − | | | | | | |
| | 7-10-30 | + + ∓ − | | | | | | |
| | 7-24-30 | + + + + | | | | | | |
| Killed | 8-7-30 | + + ∓ − | | | | | | |
| DJ3F29 Sow | 3-10-30 | + + + ∓ | | | | | Positive cultures from tissues after autopsy. Submaxillary lymph-gland + | |
| | 5-4-30 | + + + + | | | | | | |
| | 7-10-30 | + + + + | | | | | | |
| | 7-24-30 | + + + + | | | | | | |
| Killed | 8-4-30 | + + + + | | | | | | |
| DJ4S29 Sow | 11-1-29 | ∓ − − − | 11-18-29 | 3-14-30 | 10 | 0 | Placenta − | Plac. − − − − |
| | 1-14-30 | − − − − | | | | | Fetal spleen − | |
| | 3-10-30 | ± − − − | | | | | Fet. st. c. − | |
| | 5-23-30 | + + + + | | | | | | |
| | 7-10-30 | + + + + | | | | | | |
| | 7-17-30 | + + + + | | | | | | |
| DJ4F29 Sow | 3-10-30 | − − − − | | | | | Positive cultures from tissues after autopsy. Stomach contents of fetuses and placenta + | |
| | 5-23-30 | + + + + | | | | | | |
| | 7-10-30 | + + + + | | | | | | |
| | 7-17-30 | + + + + | | | | | | |
| Killed | 8-7-30 | + + + + | | | | | | |
| DJ28F28 Sow | 11-1-29 | + + + + | 11-30-29 | 3-24-30 | 6 | 2 | Fet. 1., st. c. − | Ut. s. − − − − |
| | 1-14-30 | + + ∓ ∓ | | | | | F. 2, st. c. − | St. c. − − − − |
| | 3-10-30 | ± − − − | | | | | F. 1., spleen − | Plac. − − − − |
| | 5-23-30 | + ± − − | | | | | F. 2, spleen − | |
| | 7-10-30 | + ± ∓ ∓ | | | | | Placenta − | |
| | 7-24-30 | + + ∓ ∓ | | | | | | |
| Killed | 7-30-30 | + + ± ± | | | | | | |

TABLE II—*Results of repeated agglutination tests on positive swine—Continued*

| IDENTIFICATION | DATE OF TEST | BLOOD TITRE | DATE OF BREEDING | DATE OF FARROWING | LIVE PIGS | DEAD PIGS | CULTURES | GUINEA PIG BLOOD TITRE |
|---|---|---|---|---|---|---|---|---|
| B4F29 Sow | 3-10-30 | ∓ ∓ − − | Not bred | | | | | |
| | 5-23-30 | + + + + | | | | | | |
| | 7-3-30 | + + + + | | | | | | |
| | 7-17-30 | + + + + | | | | | | |
| | 7-31-30 | + + + + | | | | | | |
| | 8-14-30 | ± ± ± ± | | | | | | |
| B6F29 Sow | 3-10-30 | + + + + | Not bred | | | | | |
| | 5-23-30 | + + + + | | | | | | |
| | 7-10-30 | + + + + | | | | | Positive cultures from tissues after autopsy. Pelvic lymph-glands + | |
| | 7-24-30 | + + + + | | | | | | |
| Killed | 8-4-30 | + + + + | | | | | | |
| B12S27 Boar | 1-18-29 | − − − − | | | | | | |
| | 4-4-29 | − − − − | | | | | | |
| | 6-10-29 | − − − − | | | | | | |
| | 11-1-29 | − − − − | | | | | | |
| | 1-14-30 | + + + + | | | | | | |
| | 3-10-30 | ± ± ± − | | | | | | |
| | 5-23-30 | + + + + | | | | | | |
| | 7-3-30 | + ± ± ± | | | | | | |
| | 7-17-30 | + ± ± ± | | | | | | |
| | 7-31-30 | + + + + | | | | | | |
| | 8-14-30 | ± ± ± ± | | | | | | |
| B15S27 Sow | 1-18-29 | − − − − | Allowed to run with boar | 8-15-29 | 7 | 1 | | |
| | 4-4-29 | − − − − | | | | | | |
| | 6-10-29 | − − − − | | | | | | |
| | 11-1-29 | − − − − | | | | | | |
| | 1-14-30 | − − − − | | | | | | |
| | 3-10-30 | ± − − − | 11-15-29 | Did not farrow | | | | |
| | 5-23-30 | + + + + | | | | | | |
| | 7-3-30 | + + + + | 3-31-30 | Did not farrow | | | | |
| | 7-17-30 | + + + + | 5-15-30 | | | | | |
| | 7-31-30 | + + + + | | | | | | |
| | 8-14-30 | + + + + | | | | | | |

TABLE II—*Results of repeated agglutination tests on positive swine—Continued*

| IDENTIFICATION | DATE OF TEST | BLOOD TITRE | DATE OF BREEDING | DATE OF FARROWING | LIVE PIGS | DEAD PIGS | CULTURES | GUINEA PIG BLOOD TITRE |
|---|---|---|---|---|---|---|---|---|
| DJ33S29 | 11-1-29 | + + + + | | | | | | |
| Sow | 1-14-30 | + + ± ∓ | | | | | | |
| | 3-10-30 | ∓ − − − | 11-28-29 | 3-24-30 | 9 | 0 | Ut. swab − | Ut. s. − − − − |
| | 5-23-30 | ∓ − − − | | | | | Positive cultures from | |
| | 7-10-30 | + ∓ − − | | | | | tissues after autopsy. | |
| | 7-24-30 | + + ± ∓ | | | | | R. submaxillary | |
| Killed | 7-30-30 | + + ∓ ∓ | | | | | lymph-gland + | |
| DJ42S29 | 11-1-29 | + + + + | | | | | | |
| Sow | 1-14-30 | + + − − | | | | | | |
| | 3-10-30 | ∓ − − − | | | | | | |
| | 5-23-30 | ∓ − − − | 2-25-30 | 6-19-30 | 11 | 1 | Fetal liver − | Ut. s. − − − − |
| | 7-10-30 | + ± ∓ − | | | | | Fetal spleen − | Placenta |
| | 7-24-30 | + + ± − | | | | | Fetal st. cont. − | |
| Killed | 8-5-30 | + ± − − | | | | | Ut. swab − | |
| | | | | | | | Placenta − | |
| | | | | | | | Positive cultures from | |
| | | | | | | | tissues after autopsy. | |
| | | | | | | | R. submaxillary | |
| | | | | | | | lymph-gland + | |
| DJ57F27 | 11-1-29 | ∓ − − − | | | | | | |
| Boar | 1-14-30 | ∓ − − − | | | | | | |
| | 3-10-30 | ∓ − − − | | | | | | |
| | 5-23-30 | + + − − | | | | | | |
| | 7-10-30 | + + + ± | | | | | Positive cultures from | |
| | 7-17-30 | + ± ∓ ∓ | | | | | tissues after autopsy. | |
| Killed | 7-28-30 | + + + + | | | | | Gall-bladder + | |
| DJ77S29 | 11-1-29 | + + + + | | | | | | |
| Sow | 1-14-30 | + − − − | | | | | | |
| | 3-10-30 | ± ∓ − − | 12-7-29 | 4-1-30 | 5 | 4 | Fetal st. c. − | Ut. s. − − − − |
| | 5-23-30 | + − − − | | | | | Fetal spleen − | St. c. − − − − |
| | 7-10-30 | + + ± ± | | | | | | |
| | 7-24-30 | + + ± ± | | | | | | |
| Killed | 8-4-30 | + + ∓ − | | | | | | |

TABLE II—*Results of repeated agglutination tests on positive swine—Continued*

| IDENTIFICATION | DATE OF TEST | BLOOD TITRE | DATE OF BREEDING | DATE OF FARROWING | LIVE PIGS | DEAD PIGS | CULTURES | GUINEA PIG BLOOD TITRE |
|---|---|---|---|---|---|---|---|---|
| DJ85F28 | 11-1-29 | + + + + | | | | | | |
| Sow | 1-14-30 | + ∓ ∓ − | | | | | | |
| | 3-10-30 | + − − − | 12-2-29 | 3-25-30 | 12 | 0 | Ut. swab + | Ut. s. − − − − |
| | 5-23-30 | + + − − | | | | | Placenta + | Plac. + + + ± |
| | 7-10-30 | + + ± − | | | | | | |
| | 7-24-30 | + + ± − | | | | | Via—G. P. | |
| Killed | 8-5-30 | ± ∓ ∓ − | | | | | Positive cultures from tissues after autopsy. Submaxillary lymph-gland + | |
| PC9F28 | 11-1-29 | + + + + | | | | | | |
| Sow | 1-14-30 | + + ∓ − | | | | | | |
| | 3-10-30 | ± ∓ − − | 11-30-29 | 3-24-30 | 10 | 0 | Placenta − | Placenta − − − − |
| | 5-23-30 | + − − − | | | | | Ut. swab − | Ut. swab − − − − |
| | 7-10-30 | + + ± − | | | | | Positive cultures from tissues after autopsy. | |
| | 7-24-30 | + + + + | | | | | Submaxillary and r. prescapular lymph-glands + | |
| Killed | 8-4-30 | + + ± ± | | | | | | |
| PC11F28 | 11-1-29 | + + ± ∓ | | | | | | |
| Sow | 1-14-30 | + + ∓ − | | | | | | |
| | 3-10-30 | + − − − | 11-30-29 | 3-26-30 | 5 | 1 | Ut. swab − | Ut. s. − − − − |
| | 5-23-30 | + ∓ ∓ − | | | | | St. cont. − | St. c. − − − − |
| | 7-10-30 | + + ± ∓ | | | | | Positive cultures from tissues after autopsy. | |
| | 7-24-30 | + + + ∓ | | | | | Kidney + | |
| Killed | 7-30-30 | + + + + | | | | | | |

TABLE II—*Results of repeated agglutination tests on positive swine—Concluded*

| IDENTIFICATION | DATE OF TEST | BLOOD TITRE | DATE OF BREEDING | DATE OF FARROWING | LIVE PIGS | DEAD PIGS | CULTURES | GUINEA PIG BLOOD TITRE |
|---|---|---|---|---|---|---|---|---|
| PC15F23 Sow | 1-18-29 | − − − − | 11-18-29 | 3-13-29 | 15 | 0 | Ut. swab − | Ut. s. − − − − |
| | 4-4-29 | − − − − | | | | | St. cont. − | St. c. − − − − |
| | 6-10-29 | − − − − | | | | | | |
| | 11-1-29 | + ∓ − − | 5-15-29 | 9-5-29 | 9 | 0 | | |
| | 1-14-30 | − − − − | | | | | | |
| | 3-10-30 | ± ∓ − − | | | | | | |
| | 5-23-30 | + + − − | 11-29-29 | 3-24-30 | 12 | 3 | Positive cultures from tissues after autopsy. R. pelvic lymph-gland + | |
| | 7-10-30 | + + ∓ − | | | | | | |
| | 7-24-30 | + ± ∓ − | | | | | | |
| Killed | 7-30-30 | + + ± ∓ | | | | | | |
| PC49F28 Sow | 11-1-29 | + + + + | 11-28-30 | 3-22-30 | 11 | 0 | Ut. swab − | Ut. s. − − − − |
| | 1-14-30 | + + + − | | | | | Placenta − | Plac. − − − − |
| | 3-10-30 | + + − − | | | | | | |
| | 5-23-30 | ∓ − − − | | | | | | |
| | 7-10-30 | + ∓ ∓ − | | | | | | |
| | 7-24-30 | + ∓ ∓ − | | | | | | |
| Killed | 7-28-30 | + ∓ − − | | | | | | |
| B329 Boar | 3-10-30 | ∓ ∓ − − | | | | | | |
| | 5-23-30 | + + + + | | | | | | |
| | 7-3-30 | + + + + | | | | | | |
| | 7-17-30 | + + + + | | | | | | |
| | 7-31-30 | + ± ± ± | | | | | | |
| | 8-14-30 | + + + + | | | | | | |

cultures obtained from uterine swabs, dead fetuses, placentas and tissues has been low.

### Blood Titre of Young Pigs Born of Positive Sows and Confined in Close Proximity to Positive Animals

After the isolation of positive reactors, eight of the positive sows farrowed in March, 1930. The sows with their litters were kept in close confinement with other positive swine in a compara-

Table III—*Blood titre of young pigs born of positive sows and confined in close proximity with positive animals*

| Identification | 5-1-30 | 6-4-30 | 7-10-30 | 7-24-30 | 8-8-30 |
|---|---|---|---|---|---|
| 1. DJ60S30 | – – – – | – – – – | – – – – | – – – – | * |
| 2. DJ59S30 | – – – – | – – – – | – – – – | – – – – | * |
| 3. DJ45S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 4. DJ46S30 | – – – – | – – – – | – – – – | ∓ – – – | – – – – |
| 5. DJ47S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 6. DJ44S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 7. DJ49S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 8. DJ50S30 | – – – – | – – – – | – – – – | + ∓ – – | – – – – |
| 9. DJ51S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 10. DJ52S30 | – – – – | – – – – | – – – – | ∓ – – – | – – – – |
| 11. DJ53S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 12. DJ54S30 | – – – – | – – – – | – – – – | ∓ – – – | – – – – |
| 13. DJ55S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 14. DJ56S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 15. DJ57S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 16. DJ40S30 | – – – – | – – – – | – – – – | – – – – | ∓ – – – |
| 17. DJ41S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 18. DJ42S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 19. DJ43S30 | – – – – | – – – – | – – – – | ∓ – – – | – – – – |
| 20. PC50S30 | – – – – | – – – – | – – – – | – – – – | ∓ – – – |
| 21. PC51S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 22. PC52S30 | – – – – | – – – – | – – – – | ∓ – – – | – – – – |
| 23. PC53S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 24. PC54S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 25. PC55S30 | – – – – * | | | | |
| 26. PC56S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 27. PC57S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 28. PC40S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 29. PC41S30 | – – – – | – – – – | – – – – | + ± ± – | + ∓ – – |
| 30. PC42S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 31. PC43S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 32. PC44S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 33. PC45S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 34. PC46S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 35. PC47S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 36. PC48S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 37. PC49S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 38. PC59S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 39. PC70S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 40. PC71S30 | – – – – | – – – – | – – – – | – – – – | * |
| 41. PC72S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 42. PC73S30 | – – – – | – – – – | – – – – | – – – – | – – – – |
| 43. PC58S30 | – – – – | – – – – | – – – – | – – – – | – – – – |

*Died.

tively small lot, in this way to see whether the young pigs would contract the disease. After these small pigs were weaned, their blood was tested five consecutive times.

A review of table III shows that the blood tests on the majority of these young swine remained negative for about four months after weaning even though they were born of mothers that were reacting positively to the agglutination test and were allowed to remain in close proximity to them. One is therefore led to believe that young swine do not become infected readily up to four months of age, at least.

### Effects of Intravenous Injection of Porcine Strain into Pregnant Sows

On April 1, 1930, four pregnant sows (Chester White 3, Chester White 4, Duroc Jersey 2 and Hampshire 25) were retained from a herd of swine in which no known abortions had occurred. These animals were tested for abortion, and their blood titres were negative in dilutions of 1:25 and above.

On April 21, they were inoculated intravenously with 1/10 slant in 1 cc of saline emulsion of *Brucella abortus* culture, porcine strain 55, previously isolated from the herd under discussion.

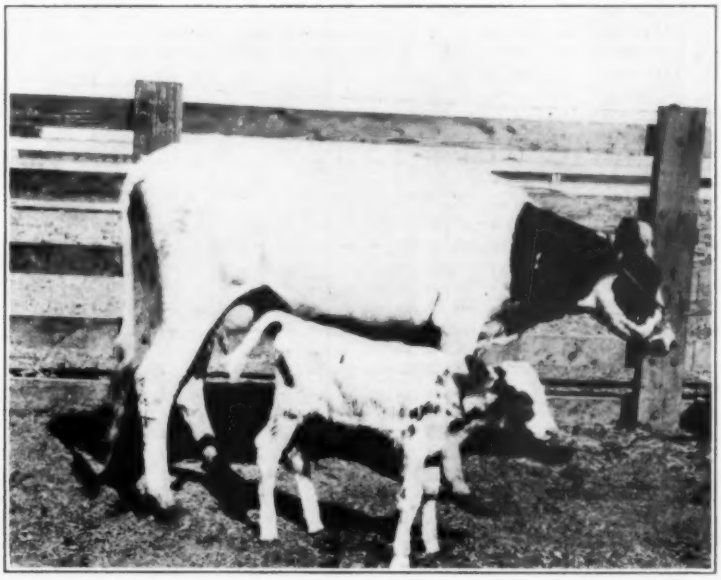

Fig. 3. Cow 116 and her calf, which reacted positively to the agglutination test, after being in close confinement with positive-reacting swine.

TABLE IV—*Results of intravenous injection of porcine strain into pregnant sows*

| Identification | Date of Inoculation | Material and Method of Inoculation | Date of Breeding | Date of Farrowing and No. in Litter | Length of Pregnancy | Date of Blood Test | Result of Agg. Test | Cultures | Guinea Pig Cultures | Guinea Pig Blood Titre | Cultures of Tissues after Post Mortem and Date |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Sow 2 (Duroc-Jersey) | 4-21-30 | 1/10 slant in 1 cc saline emul. *Br. abortus* cult. porcine strain 55 injected in ear vein | 1-20-30 | Not known. Fetuses not found | Not known | 4-2-30 | − − − − | Uterine swab − | Ut. swab + | + + + + | |
| | | | | | | 4-29-30 | + + + + + ∓ − − | | Ut. swab + | + + + + | Still living |
| | | | | | | 5-6-30 | + + + + + + ± − − | | G.P. lesions: | | |
| | | | | | | 5-13-30 | + + + + + + ∓ − − | | Liver + | | |
| | | | | | | 5-20-30 | + + + + + + ± − − | | Spleen + | | |
| | | | | | | 5-27-30 | + + + + + + ± − − | | Cultures of | | |
| | | | | | | 6-3-30 | + + + + + + ∓ − − | | above + | | |
| | | | | | | 6-10-30 | + + + + + + + − − | | | | |
| | | | | 7-29-30 | + + + + + ∓ ∓ − − | 6-17-30 | + + + + + + − − − | | | | |
| | | | | 8-5-30 | + + + + + + ∓ − − | 6-24-30 | + + + + + ∓ − − − | | | | |
| | | | | | | 7-1-30 | + + + + + + ± − − | | | | |
| | | | | | | 7-8-30 | + + + + + ± | | | | |
| | | | | | | 7-15-30 | + + + + + + ∓ − − | | | | |
| | | | | | | 7-22-30 | + + + + + + ± − − | | | | |
| Sow 25 (Hampshire) | 4-21-30 | 1/10 slant in 1 cc saline emul. *Br. abortus* cult. porcine strain 55 injected in ear vein | 1-13-30 | 7-5-30 5 alive 1 dead | Full time | 4-2-30 | − − − − | Fet. spl. − | St. cont. − | Died | Killed 7-26-30 |
| | | | | | | 4-29-30 | + + + + + ± − | St. cont. − | St. cont. − | − − − − | Rt. supra-mammary lymph-gland + |
| | | | | | | 5-6-30 | + + + + + + ± ∓ − | Placenta − | No lesions | | |
| | | | | | | 5-13-30 | + + + + + ∓ ∓ − − | Ut. swab − | | | |
| | | | | | | 5-20-30 | + + + + + ∓ − − − | | Ut. swab + | + + + + | |
| | | | | | | 5-27-30 | + + + + ± ∓ − − − | | Ut. swab + | + + + + | |
| | | | | | | 6-3-30 | + + + + + ∓ − − − | | Placenta | Died | L. supra-mammary lymph-gland + |
| | | | | | | 6-10-30 | + + + + + ± − − − | | Placenta + | + + + + | |
| | | | | | | 6-17-30 | + + + + ± − − − − | | | | |
| | | | | | | 6-24-30 | + + + + + − − − − | | | | |
| | | | | | | 7-1-30 | + + + + + + ∓ − − | | | | |
| | | | | | | 7-8-30 | + + + + + ∓ − − − | | | | |
| | | | | | | 7-15-30 | + + + + + + ∓ − − | | | | |
| | | | | | | 7-22-30 | + + + + + + ∓ − − | | | | |
| | | | | | | 7-26-30 | + + + + + + ± − − | | | | |
| Sow 4 (Chester White) | 4-21-30 | 1/10 slant in 1 cc saline emul. *Br. abortus* cult. porcine strain 55 injected in ear vein | 1-16-30 | 5-10-30 | Full time | 4-2-30 | − − − − | Ut. swab − | Ut. swab + | + + + + | Died Jun. 20 |
| | | | | | | 4-29-30 | + + + ∓ − | Ut. swab − | Ut. swab + | + + + + | L. iliac − |
| | | | | | | 5-6-30 | + + + + + + ± ∓ − | | | | R. prescap. − |
| | | | | | | 5-13-30 | + + + + + + ∓ ∓ − | | | | L. prescap. − |
| | | | | | | 5-20-30 | + + + + ∓ − − − − | | | | R.precruc. − |
| | | | | | | 5-27-30 | + + + + ∓ − − − − | | | | L. precruc. − |
| | | | | | | 6-3-30 | + + + ± − − − − − | | | | R.submax. − |
| | | | | | | 6-10-30 | + + + ± − − − − − | | | | Mammary − |
| | | | | | | 6-17-30 | + + + ∓ − − − − − | | | | Spleen − |
| | | | | | | | | | | | Urine − |
| | | | | | | | | | | | Ut. r. horn − |
| | | | | | | | | | | | Ut. l. horn + |

TABLE IV—*Results of intravenous injection of porcine strain into pregnant sows—Concluded*

| Identification | Date of Inoculation | Material and Method of Inoculation | Date of Breeding | Date of Farrowing and No. in Litter | Length of Pregnancy | Date of Blood Test | Result of Agg. Test | Cultures | Guinea Pig Cultures | Guinea Pig Blood Titre |
|---|---|---|---|---|---|---|---|---|---|---|
| Sow No. 3 Chester White | 4-21-30 | 1/10 slant in 1 cc saline emul. *Br. Abortus* cult. porcine strain 55 injected in ear vein | 1-9-30 | 5-3-30<br>7 alive<br>2 dead | Full time | 4-2-30<br>4-29-30<br>5-6-30<br>5-13-30<br>5-20-30<br>5-27-30<br>6-3-30 | – – – –<br>+ + + + + ∓ – –<br>+ + + + + + ∓ –<br>+ + + + + ± –<br>+ + + + + ± –<br>+ + + + + ∓ –<br>+ + + + + ∓ – | F. l., spl. –<br>F. st. cont. +<br>F. l. spl. –<br>F. st. cont. + | St. cont. –<br>St. cont. –<br>Ut. swab. +<br>Ut. swab +<br>Placenta –<br>Placenta + | Died<br>– – – –<br>+ + + +<br>+ + + +<br>Died<br>+ + + + |

*Cultures of tissues after postmortem, killed June 3, 1930*

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Supramam. | + | Periaor. | + | Mesentery | + | Mammary | + | Lym. n. r. h. | | Urine | – | H. ut. | + |
| Sternal | + | R. iliac | + | L. horn ut. | + | Spleen | + | Uterus | + | L. iliac | – | | |
| R. submax. | + | Portal | + | R. horn ut. | + | Liver | + | Bone marrow | – | L. ovary | – | | |
| L. submax. | + | R. inguinal | + | R. ovary | + | Suprarenal | + | Kidney | – | Lym. n. l. | – | | |

A review of table IV shows that these four sows did not abort but carried their pigs full time. The blood titre of these animals has remained uniformly high. Positive cultures of the organism were obtained from the uterine swabs and placentas. The blood titre of the inoculated guinea pigs was uniformly high. Tissues cultured from three of these swine after autopsy showed that the organisms were widely distributed in one (sow 3) and found only in the uterine horn and mammary gland, respectively, of two others.

TABLE V—*Breeding and farrowing record of positive and negative sows from January, 1929, to August, 1930*

| POSITIVE | | | | | | NEGATIVE | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| IDENTIFICATION | GESTATION (IN DAYS) | ALIVE | | DEAD | | IDENTIFICATION | GESTATION (IN DAYS) | ALIVE | | DEAD | |
| | | No. | % | No. | % | | | No. | % | No. | % |
| DJ1F26 | 113 | 9 | 75.0 | 3 | 25.0 | B15S27 | 114 | 7 | 87.5 | 1 | 12.5 |
| DJ4S29 | 114 | 10 | 76.9 | 3 | 23.1 | B38F23 | 112 | 5 | 62.5 | 3 | 37.5 |
| DJ5S28 | 73 | 0 | 0.0 | 8 | 100.0 | | | | | | |
| DJ6S27 | 115 | 6 | 100.0 | 0 | 0.0 | DJ10S24 | 103 | 0 | 0.0 | 10 | 100.0 |
| DJ10S29 | 42 | 0 | 0.0 | 9 | 100.0 | DJ31F26 | 96 | 0 | 0.0 | 9 | 100.0 |
| DJ19P | 112 | 12 | 92.3 | 1 | 7.7 | DJ35S28 | 113 | 9 | 60.0 | 6 | 40.0 |
| DJ24S26 | 113 | 9 | 100.0 | 0 | 0.0 | DJ52F27 | 114 | 4 | 80.0 | 1 | 20.0 |
| DJ28F28 | 114 | 6 | 75.0 | 2 | 25.0 | | 114 | 5 | 100.0 | 0 | 0.0 |
| DJ32F26 | 115 | 6 | 75.0 | 2 | 25.0 | | 114 | 6 | 66.7 | 3 | 33.3 |
| | 81 | 0 | 0.0 | 9 | 100.0 | DJ55F28 | 115 | 8 | 88.9 | 1 | 11.1 |
| DJ33S29 | 112 | 9 | 100.0 | 0 | 0.0 | DJ57T | 115 | 9 | 100.0 | 0 | 0.0 |
| DJ42S29 | 114 | 11 | 91.7 | 1 | 8.3 | DJ59F27 | 117 | 9 | 81.8 | 2 | 18.2 |
| DJ45F27 | 92 | 0 | 0.0 | 9 | 100.0 | DJ64S23 | 113 | 10 | 66.7 | 5 | 33.3 |
| DJ55S25 | 109 | 6 | 30.0 | 14 | 70.0 | | 114 | 9 | 69.2 | 4 | 30.8 |
| DJ61F27 | 117 | 11 | 84.6 | 2 | 15.4 | | 114 | 9 | 75.0 | 3 | 25.0 |
| DJ66F28 | 115 | 0 | 0.0 | 1 | 100.0 | DJ79F28 | 114 | 7 | 100.0 | 0 | 0.0 |
| DJ77S29 | 115 | 5 | 55.5 | 4 | 44.5 | | | | | | |
| DJ85F28 | 113 | 12 | 100.0 | 0 | 0.0 | PC8S29 | 111 | 5 | 83.3 | 1 | 16.7 |
| DJ136S28 | 115 | 4 | 57.1 | 3 | 42.9 | PC10S19 | 119 | 5 | 83.3 | 1 | 16.7 |
| | | | | | | | 123 | 0 | 0.0 | 2 | 100.0 |
| PC9F28 | 114 | 10 | 100.0 | 0 | 0.0 | PC10F28 | 114 | 9 | 100.0 | 0 | 0.0 |
| PC10S27 | 114 | 2 | 50.0 | 2 | 50.0 | PC15F23 | 117 | 15 | 100.0 | 0 | 0.0 |
| PC11F28 | 115 | 5 | 83.3 | 1 | 16.7 | PC20S29 | 116 | 10 | 100.0 | 0 | 0.0 |
| PC15F23 | 114 | 9 | 100.0 | 0 | 0.0 | PC21F20 | 113 | 6 | 85.7 | 1 | 14.3 |
| | 115 | 12 | 80.0 | 3 | 20.0 | PC41S28 | 117 | 9 | 100.0 | 0 | 0.0 |
| PC16S23 | 117 | 6 | 66.7 | 3 | 33.3 | PC84S28 | 115 | 1 | 100.0 | 0 | 0.0 |
| PC16F27 | 120 | 4 | 100.0 | 0 | 0.0 | | | | | | |
| PC21S29 | 115 | 5 | 100.0 | 0 | 0.0 | | | | | | |
| PC22S25 | 114 | 9 | 81.8 | 2 | 18.2 | | | | | | |
| PC30 | 117 | 5 | 100.0 | 0 | 0.0 | | | | | | |
| PC33F26 | 117 | 7 | 100.0 | 0 | 0.0 | | | | | | |
| PC49F28 | 114 | 11 | 100.0 | 0 | 0.0 | | | | | | |
| 29 sows 31 litters | | 201 | | 82 | | 19 sows 24 litters | | 157 | | 53 | |
| Average per litter | | 6.48 | 71.02 | 2.64 | 28.98 | | | 6.54 | 74.76 | 2.21 | 25.24 |

### Breeding and Farrowing Record of Positive and Negative Reacting Sows from January, 1929 to August, 1930

To obtain a more definite comparison between the breeding and farrowing records of positive and negative sows in this herd, the results have been tabulated in table V.

A review of table V shows that of 31 litters from 29 positive sows, 201 pigs were born alive and 82 were born dead, or an average of 6.48 live pigs per litter and 2.64 dead pigs per litter. The time of gestation was normal for all animals except five. Sow DJ 10 S 29 aborted 9 dead pigs at 42 days; Sow DJ 5 S 28 aborted 8 dead pigs at 81 days; and Sow DJ 55 S 25 aborted 14 dead and 6 live pigs at 109 days.

The negative swine included 19 sows having 24 litters. All animals farrowed normally except one. Sow DJ 31 F 26 aborted 9 dead pigs at 96 days. The cultures from these fetuses and placentae were negative, and the sow's blood titre has remained negative to the agglutination test. The average percentage of pigs to the litter was 6.54 live and 2.21 dead pigs.

### Blood Titre of Pigs of a Positive Sow before and After Receiving Colostrum and after Weaning

A Duroc Jersey sow (42 S 29), reacting positively to the agglutination test, farrowed 12 pigs (one dead and 11 alive) on June 19, 1930. Blood was obtained from these pigs before they received any colostrum, and their blood was negative to the agglutination test. Five days later (June 24, 1930), after they had received colostrous milk from the sow, another blood test

Table VI—*Blood titre of pigs of a positive sow before and after receiving colostrum and after weaning*

| Farrowed | Pig | Blood Titre before Receiving Colostrum | Date of Test after Receiving Colostrum | Blood Titre after Receiving Colostrum | Date of Test after Weaning | Blood Titre after Weaning |
|---|---|---|---|---|---|---|
| | 1 | − − − − | | + + + − | | − − − − |
| | 2 | − − − − | | + + + ± | | − − − − |
| | 3 | − − − − | | + ∓ − − | | − − − − |
| | 4 | − − − − | | + + ± − | | − − − − |
| | 5 | − − − − | | + + + − | | − − − − |
| 6-19-30 | 6 | − − − − | 6 -24-30 | + ± ∓ − | 8-2-30 | − − − − |
| | 7 | − − − − | | + + ± − | | − − − − |
| | 8 | − − − − | | + ± − − | | − − − − |
| | 9 | − − − − | | + + ∓ − | | − − − − |
| | 10 | − − − − | | + + ∓ ∓ | | − − − − |
| | 11 | − − − − | Died | | | |

was taken and all pigs reacted positively to the agglutination test. A third blood test was taken after weaning (August 2, 1930), and the blood titre of these small pigs had become negative.

### Transmission of Porcine Strain to a Cow by Contact With Swine

March 18, 1930, a Holstein cow, which had a history of coming from a negative herd and reacting negatively to the agglutination test, was available and as a preliminary experiment was placed in a small enclosure with twelve reacting sows, eight of which farrowed during this time. Certain results are shown in table VII.

Table VII—*Result of close contact between negative cow and positive swine*

| Animal | Born | Breeding | Date of Tests | Blood Titre | Calving Record | Cultures |
|---|---|---|---|---|---|---|
| Cow 116 | 11-16-26 | Sire 1970 Dam 2305 | 10-27-27 (Tested frequently) | − − − − − − − − | Normal 10-27-28 | Negative |
| Placed with positive reacting swine (natural infection), March 18, 1930. | | | | | | |
| | | | 4-18-30 | − − − − | | |
| | | | 5-19-30 | − − − − | | |
| | | | 6-3-30 | − − − − | | |
| | | | 7-22-30 | + + ∓ ∓ − | | |
| | | | 8-5-30 | + ± ∓ ∓ − | Normal (Heifer) | Whey Agg. + + + ∓ ∓ − |
| | | | 8-15-30 | ± ± ± ∓ − | 8-12-30 (Calf killed) 8-15-30 Blood titre ± ± − − | |
| | Killed 8-15-30 | | | | | |

Table VII shows that this cow (116) was in close confinement with twelve positive sows and their offspring for approximately three months before reacting positively to the agglutination test. This result is significant from the standpoint of both cattle and human infection, since the swine strain has shown itself to be more virulent for man.

### Summary

Incomplete isolation of swine reacting as positive and suspicious to the agglutination test was not of much value, since each consecutive blood test showed new reacting animals.

The blood titre of reacting swine is variable and appears to recede at farrowing and rise again to its previous high level.

A group of forty-three pigs born of positive sows and confined in close quarters with positive animals reacted negatively for approximately three months after weaning, when one animal (29) reacted positively, one (8) suspiciously, and five animals (4, 10, 12 and 22) reacted in 1:25 dilution only.

Four sows advanced in pregnancy were inoculated intravenously with 1/10 slant of *Brucella abortus* (suis), U. C. culture 55. These animals farrowed at full time; their blood titre remained uniformly high. The tissue cultures from one of these swine after being killed showed that the organisms were well distributed throughout the body.

The breeding record of positive and negative sows from January, 1929, to August, 1930, shows an average percentage litter of 6.48 live pigs, and 2.64 dead pigs from the positive sows. The negative sows had a percentage litter of 6.54 live and 2.21 dead pigs.

The time of gestation for the positive sows (31 farrowing) was normal, with the exception of five sows which aborted, one at 42 days, one at 73 days, one at 92 days, one at 81 days, and one at 109 days.

The blood titre of young swine at birth, before they obtained colostrum, was negative.

Blood tests taken on young swine, after they had colostrous milk from positive sows, reacted positively to the agglutination test but again reacted negatively after weaning.

A negative Holstein cow (116) reacted positively to the agglutination test after 91 days close contact with positively reacting sows and their offspring.

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## Oklahoma Needs Veterinarians

Dr. Walter H. Martin, secretary of the Oklahoma State Board of Veterinary Medical Examiners, is authority for the statement that there are fifteen towns in Oklahoma, with populations ranging from 2,000 to 10,000 without a graduate veterinarian. Dr. Martin says Oklahoma needs qualified men of good character.

# NATURAL BRUCELLA INFECTION IN SWINE*

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Since Brucella was isolated by Traum,[1] from fetuses expelled prematurely from sows, the belief has been widely prevalent that Brucella disease in swine is essentially an abortion disease. In general, this belief is based on the sporadic occurrence of abortion in swine and confirmatory experiments which have been conducted with the object in view of producing premature expulsion of fetuses. Our studies of the natural course of the disease in several large groups of hogs in the state of Michigan have failed to confirm this belief. In other words, the data which we have accumulated show that Brucella infection in swine is not essentially an abortion disease.

The natural occurrence of abortions in hogs due to the porcine species of Brucella, or its natural transmissibility from sow to boar or from boar to sow, has been conclusively demonstrated by Traum,[1] Good and Smith,[2] Hayes and Traum,[3] Doyle and Spray,[4] Beach,[5] Hadley,[6] Connaway, Durant and Newman,[7,8] Graham, Boughton and Tunnicliff,[9,10] Smith[11] and many others.

Assuming that the naturally infected hog is the chief source of the species of Brucella known as *Brucella suis*, the important role that it is now playing as a cause of Brucella infection in humans, aside from that in animals, makes it imperative that more should be learned about the natural infection in hogs. In learning more about the course of the natural disease in hogs and its nature, it is obvious that one will be in a better position to proceed with, and make recommendations for, the prevention of the disease in hogs and at the same time circumvent infection in humans.

Early this year an opportunity was presented for collection of data on the course of the disease in a large herd of hogs in which Brucella infection was known to have been present, on the basis of serological tests, as early as 1923. We are, therefore, presenting these data and some results of a field survey in this report.

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The discussion in this report concerns only that which has a bearing on natural Brucella infection in swine.

## Method of Study

*Serological examinations:* When blood specimens from the hogs were desired, tail-bleeding was resorted to. Sufficient blood for examination may be collected in this way in a very short time. The rapid agglutination method for detecting Brucella agglutinins in serum was used throughout the study. Occasionally, it was compared with the test-tube method for accuracy. The results obtained by the two methods were always in agreement. The amounts of serum used in titrating each blood specimen for agglutinins were 0.08, 0.04, 0.02, 0.01 and 0.004 cc. These respective amounts of serum represent dilutions of 1:25, 1:50, 1:100, 1:200 and 1:500. The following signs describe the reactions obtained: —, negative; T, trace of agglutination; P, incomplete agglutination; +, complete agglutination. In tables where the dilution figures are not placed at the top of the columns containing the serological reactions, the amounts of serum used are the same as those just mentioned.

*Bacteriological examinations:* When a hog was slaughtered, pieces of tissue were taken from each of the internal organs and the muscles. The skeletal and visceral lymph-nodes were removed from all parts of the carcass. Pieces of the tissues and nodes were smeared over the surface of several beef liver-agar plates. The inoculated medium was then incubated both aerobically and in an increased atmosphere of $CO_2$ at 37° C. for seven days before discarding. Tissue from the organs of aborted or still-born fetuses were cultured in the same manner. In the case of fetal membranes, portions of them were removed and ground to a pulp with fine sand and saline solution in a mortar. About 0.5 to 1.0 cc of the macerated tissue was then injected subcutaneously into a guinea pig. After a lapse of six weeks, the pig was sacrificed and examined for the presence of the organism.

All cultures isolated were identified by means of the agglutination test and the dye media differential method.[12]

## Results

At the beginning of this study, there were 180 young and mature hogs in this one particular herd. The herd was composed of the following breeds: Yorkshire, Berkshire, Hampshire, Chester, Duroc, Poland China and Tamworth. There were 48 sows bred in the fall of 1929, to farrow in March and April of 1930.

TABLE I—*Agglutination reactions of hogs showing Br. suis at time of slaughter*

| HOG | BREED | SEX | DATE AND TITRE OF AGGLUTINATION TEST | | | | KILLED | TITRE | BACTERIOLOGICAL FINDINGS (LOCATION OF *Br. Suis*) |
|---|---|---|---|---|---|---|---|---|---|
| | | | 1–6–30 | 2–17–30 | 4–3–30 | 6–9–30 | | | |
| 1 | Yorkshire | Gilt | + + + + + | + + + + + | + P T – – | + + + + + | 7– 7–30 | + + + + + | Spleen, gastric and internal iliac node |
| 2 | Poland China | Sow | + + P T – | + + + + + | | | 5– 2–30 | + + + + + | Spleen, gastric and supra-mammary nodes |
| 3 | Yorkshire | Sow | + + + + + | | | | 1–23–30 | + + + + + | Uterus |
| 4 | Berkshire | Sow | + + + P T | | | | 1–23–30 | + + + P T | Mammary glands and spleen |
| 5 | Hampshire | Sow | P T – – – | | | | *1–13–30 | P T – – – | Spleen, liver and lymph-nodes |
| 6 | Yorkshire | Barrow | – – – – – | + P T – – | | | 2–20–30 | + P T – – | Spleen |
| 7 | Chester | Sow | P P T – – | + P T – – | | | 2–27–30 | + P T – – | Gastric lymph-nodes |
| 8 | Yorkshire | Gilt | + + + P P | | | | 3–10–30 | + + P T – | Spleen |
| 9 | Duroc | Sow | P T – – – | + + + + + | | | 3–13–30 | + + P T – | Spleen, kidneys and sub-maxillary nodes |
| 10 | Duroc | Sow | + + + P T | + P T – – | | | 4–12–30 | + + + + + | Kidney, gastric and supra-mammary nodes |
| 11 | Tamworth | Sow | + + + + + | + + + P T | + + + P T | | 4–12–30 | + + + P T | Spleen |
| 12 | Poland China | Sow | P T – – – | + + P T – | | | 3– 3–30 | + + P T – | Spleen |
| 13 | Chester | Sow | T – – – – | + + + P T | | | | + + + P T | Spleen, submaxillary and supramammary nodes |
| 14 | Chester | Sow | + + + + + | | + + P T – | + + P T – | 6–27–30 | + + P T – | Spleen |
| 15 | Poland China | Sow | + + + P T | | | | 3–31–30 | + + + + P | Supramammary nodes |

*Died.

During the winter months, the hogs were housed in small pens in a building. When the weather became warmer, they were kept in feed-lots, usually in small groups. At the time of farrowing, each sow was placed in an inclosure separate from the other hogs.

The sanitary conditions which prevailed in the hog-barn and lots were excellent, and superior to those one usually encounters on the average hog-farm.

Many of the hogs were sold out of the herd, to be slaughtered for pork and for breeding purposes each year. In fact, rarely were brood sows, barrows or boars ever kept on the premises for more than two years.

There has never been any considerable loss from premature expulsion of fetuses in sows in this herd, but there have been many occurrences in the past of the delivery of still-born pigs at farrowing time. Also, at times, considerable difficulty was encountered in getting sows to conceive, but the number involved each year was small in comparison to the number that conceived.

The first agglutination test conducted on this herd in 1930 revealed 18 hogs reacting in a titre of 1:100 or above and 28 in titres below 1:100. In other words, the test gave evidence that 46 animals (25.5 per cent) in the herd had at least been exposed to Brucella. Criticism might be raised of our interpreting reactions as low as a trace in a 1:25 dilution as meaning exposure to Brucella infection, but subsequent tests and bacteriological findings revealed that low reactions usually have some significance.

For the purpose of clarity, the hogs studied have been divided into four groups and the data concerning them recorded in three separate tables, namely: table I, those slaughtered and showing *Br. suis* in their tissues; table II, those slaughtered and not showing *Br. suis* in their tissues; table III, those not yet slaughtered. The fourth group is comprised of those gilts and sows that have never reacted to the agglutination test and are still in the herd.

In table I are recorded the agglutination test records and bacteriological findings on 15 hogs from which *Br. suis* was recovered at the time of slaughter or death. Of the total, 13 were bred to farrow in the spring of 1930. Nine of these failed to farrow, due either to their failure to conceive or early unobserved abortions, and one aborted near the time of farrowing. The fetal membranes of 4 sows (12, 13, 14 and 15) were examined for the presence of *Br. suis*, but in only one case (sow 13) was it found. This sow happened to be the only one in the entire herd that aborted during this breeding season. She was bred December 6

TABLE II—*Agglutination reactions of hogs not showing Br. suis at time of slaughter*

| HOG | BREED | SEX | DATE AND TITRE OF AGGLUTINATION TEST | | | | | | | | | | | | | | | KILLED | TITRE | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | 1-6-30 | | | | | 2-17-30 | | | | 4-3-30 | | | | 6-7-30 | | | | | | | |
| 1 | Tamworth | Sow | – | – | – | – | – | + | P | T | – | – | P | T | – | – | – | P | T | – | – | – | 7- 7-30 | P | T | – | – | – |
| 2 | Duroc | Sow | + | + | P | T | – | + | P | P | T | – | + | P | P | T | – | | | | | | 5- 2-30 | + | P | P | T | – |
| 3 | Yorkshire | Gilt | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 7- 7-30 | – | – | – | – | – |
| 4 | Poland China | Sow | P | P | T | – | – | + | P | T | – | – | | | | | | | | | | | 5- 2-30 | + | P | T | – | – |
| 5 | Yorkshire | Sow | P | P | T | – | – | + | P | T | – | – | P | P | T | – | – | P | P | T | – | – | 7- 7-30 | P | P | T | – | – |
| 6 | Tamworth | Sow | + | + | P | T | – | + | + | + | + | + | | | | | | + | + | + | + | P | 7- 7-30 | + | P | T | – | – |
| 7 | Berkshire | Gilt | + | + | + | + | + | + | + | + | + | P | | | | | | | | | | | 4-12-30 | + | + | P | T | – |
| 8 | Duroc | Sow | P | T | – | – | – | + | + | + | P | T | | | | | | – | – | – | – | – | 7- 7-30 | – | – | – | – | – |
| 9 | Berkshire | Sow | T | – | – | – | – | + | + | + | P | T | – | – | – | – | – | – | – | – | – | – | 7- 7-30 | – | – | – | – | – |
| 10 | Poland China | Sow | – | – | – | – | – | + | + | + | + | P | P | T | – | – | – | P | T | – | – | – | 7- 7-30 | P | T | – | – | – |
| 11 | Berkshire | Sow | + | P | T | – | – | P | T | – | – | – | P | P | T | – | – | P | T | – | – | – | 7- 7-30 | – | – | – | – | – |
| 12 | Chester | Sow | + | + | P | T | – | + | + | + | + | + | + | + | P | T | – | T | – | – | – | – | 7- 7-30 | T | – | – | – | – |
| 13 | Duroc | Sow | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 7- 7-30 | – | – | – | – | – |
| 14 | Duroc | Sow | – | – | – | – | – | P | T | – | – | – | | | | | | + | P | T | – | – | 7- 7-30 | P | T | – | – | – |
| 15 | Duroc | Gilt | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 7- 7-30 | – | – | – | – | – |
| 16 | Duroc | Gilt | – | – | – | – | – | T | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 7- 7-30 | – | – | – | – | – |
| 17 | Yorkshire | Gilt | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | + | + | + | P | T | 7- 7-30 | + | P | T | – | – |
| 18 | Yorkshire | Barrow | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | | | | | | 5- 2-30 | – | – | – | – | – |
| 19 | Yorkshire | Barrow | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | | | | | | 5- 2-30 | – | – | – | – | – |
| 20 | Yorkshire | Gilt | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | | | | | | 5- 2-30 | – | – | – | – | – |
| 21 | Chester | Sow | – | – | – | – | – | + | + | + | P | T | + | + | P | T | – | + | + | P | T | – | 7- 7-30 | + | + | + | P | T |
| 22 | Yorkshire | Barrow | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | | | | | | 5- 2-30 | – | – | – | – | – |
| 23 | Berkshire | Gilt | + | P | T | – | – | – | – | – | – | – | – | – | – | – | – | | | | | | 5- 2-30 | – | – | – | – | – |
| 24 | Berkshire | Gilt | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | | | | | | 5- 2-30 | – | – | – | – | – |
| 25 | Berkshire | Gilt | + | + | + | P | T | + | + | + | + | P | + | + | + | + | + | P | P | T | – | – | 7- 7-30 | – | – | – | – | – |
| 26 | Berkshire | Gilt | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | P | P | T | – | – | 7- 7-30 | – | – | – | – | – |
| 27 | Yorkshire | Barrow | – | – | – | – | – | – | – | – | – | – | | | | | | | | | | | 4-12-30 | – | – | – | – | – |
| 28 | Berkshire | Sow | – | – | – | – | – | – | – | – | – | – | | | | | | | | | | | 4-12-30 | – | – | – | – | – |
| 29 | Berkshire | Gilt | + | P | T | – | – | – | – | – | – | – | | | | | | | | | | | 4-12-30 | – | – | – | – | – |
| 30 | Yorkshire | Barrow | – | – | – | – | – | – | – | – | – | – | | | | | | | | | | | 5- 2-30 | – | – | – | – | – |
| 31 | Yorkshire | Gilt | P | T | – | – | – | – | – | – | – | – | | | | | | | | | | | 5- 2-30 | – | – | – | – | – |
| 32 | Yorkshire | Barrow | – | – | – | – | – | T | – | – | – | – | | | | | | | | | | | 5- 2-30 | – | – | – | – | – |
| 33 | Yorkshire | Barrow | – | – | – | – | – | – | – | – | – | – | | | | | | | | | | | 5- 2-30 | – | – | – | – | – |
| 34 | Yorkshire | Gilt | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | | | | | | 6- 9-30 | – | – | – | – | – |
| 35 | Berkshire | Gilt | P | T | – | – | – | – | – | – | – | – | | | | | | | | | | | 5- 2-30 | – | – | – | – | – |
| 36 | Berkshire | Gilt | – | – | – | – | – | – | – | – | – | – | | | | | | | | | | | 5- 2-30 | – | – | – | – | – |
| 37 | Berkshire | Gilt | – | – | – | – | – | – | – | – | – | – | | | | | | | | | | | 6- 9-30 | – | – | – | – | – |
| 38 | Berkshire | Sow | + | + | + | P | T | | | | | | P | T | – | – | – | T | – | – | – | – | 6-27-30 | T | – | – | – | – |
| 39 | Tamworth | Sow | + | + | + | + | P | T | – | – | – | – | – | – | – | – | – | | | | | | 6-27-30 | – | – | – | – | – |

1930, and aborted six fetuses, March 21, 1930. *Br. suis* was recovered in pure culture from all of the internal organs of each fetus

The bacteriological findings in the hogs would indicate that the spleen is the proper organ to examine in searching for *Br. suis*, as it was recovered from 11 of the 15 spleens examined. The lymph-nodes, both skeletal and visceral, are next in importance as locations for *Br. suis*. It is interesting to note that the organism was recovered from the udder in only one case (sow 4). These findings would indicate that the organism does not localize in the udder of hogs to the same extent as *Br. abortus* (Bang) does in the udders of cattle.

In table II are set forth the agglutination test records of those hogs from which *Br. suis* was not recovered at the time of slaughter.

Of the 30 sows and gilts in this group, 17 (1, 2, 3, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 21, 28, 38 and 39) farrowed during the spring of 1930. Sow 4 was bred, but failed to farrow, due to failure to conceive or early unobserved abortion.

Many of these hogs failed to show even a trace of agglutinins in their blood during the period of study. The 24 hogs in this group that reacted represent 61 per cent of the total number of reacting hogs which were slaughtered. It may be noted that there was a tendency for the agglutination titres to disappear in the entire group with one exception (sow 21).

*Br. suis* was not recovered from the tissues of these hogs, possibly because they had recovered from infection before they were slaughtered, as indicated by a fall in the agglutination titre in most cases.

In table III are set forth the serological findings over a 9-month period on 23 hogs that have not yet been slaughtered. There are 6 boars in this group, from 6 months to 2 years of age. Of the 17 sows and gilts, 12 were bred and farrowed in the spring of 1930.

It may be noted from the table that the agglutination titres failed to become very high in most instances. Most of the hogs have now ceased to react.

## Survey of Brucella Infection in Other Swine Herds in Michigan

In the light of our findings in the herd of hogs which the foregoing data concern, we thought it might prove profitable to ascertain the extent of infection in other herds in various parts

TABLE III—*Trend of agglutination titre in hogs not yet slaughtered*

| Hog | Breed | Sex | Jan., 1930 | | | | | Feb., 1930 | | | | | April, 1930 | | | | | June, 1930 | | | | | July, 1930 | | | | | Aug., 1930 | | | | | Sept., 1930 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | 1:25 | 1:50 | 1:100 | 1:200 | 1:500 | 1:25 | 1:50 | 1:100 | 1:200 | 1:500 | 1:25 | 1:50 | 1:100 | 1:200 | 1:500 | 1:25 | 1:50 | 1:100 | 1:200 | 1:500 | 1:25 | 1:50 | 1:100 | 1:200 | 1:500 | 1:25 | 1:50 | 1:100 | 1:200 | 1:500 | 1:25 | 1:50 | 1:100 | 1:200 | 1:500 |
| 1 | Poland China | Boar | + | + | P | T | – | + | + | + | P | T | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| 2 | Duroc | Boar | T | – | – | – | – | P | T | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| 3 | Yorkshire | Sow | P | T | – | – | – | – | – | – | – | – | P | T | – | – | – | – | – | – | – | – | + | P | T | – | – | – | – | – | – | – | – | – | – | – |
| 4 | Duroc | Sow | – | – | – | – | – | P | T | – | – | – | T | – | – | – | – | P | T | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| 5 | Poland China | Gilt | – | – | – | – | – | P | T | – | – | – | T | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| 6 | Poland China | Gilt | – | – | – | – | – | – | – | – | – | – | T | – | – | – | – | + | P | T | – | – | T | – | – | – | – | – | – | – | – | – | – | – | – | – |
| 7 | Yorkshire | Sow | T | – | – | – | – | P | T | – | – | – | P | T | – | – | – | + | P | T | – | – | T | – | – | – | – | P | T | – | – | – | P | T | – | – | – |
| 8 | Chester | Gilt | – | – | – | – | – | P | T | – | – | – | P | T | – | – | – | P | T | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| 9 | Hampshire | Sow | P | T | – | – | – | P | T | – | – | – | P | T | – | – | – | T | – | – | – | – | P | T | – | – | – | P | T | – | – | – | P | T | – | – | – |
| 10 | Yorkshire | Sow | P | P | T | – | – | + | P | T | – | – | P | P | T | – | – | P | P | T | – | – | P | P | T | – | – | | | | | | | | | | |
| 11 | Poland China | Sow | – | – | – | – | – | + | P | T | – | – | T | – | – | – | – | – | – | – | – | – | P | T | – | – | – | | | | | | | | | | |
| 12 | Yorkshire | Sow | – | – | – | – | – | – | – | – | – | – | P | T | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| 13 | Chester | Sow | + | + | P | T | – | P | P | T | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| 14 | Chester | Gilt | + | + | + | + | P | + | + | + | + | P | P | T | – | – | – | – | – | – | – | – | T | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| 15 | Berkshire | Sow | + | + | + | + | P | + | + | P | T | – | P | T | – | – | – | P | P | T | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| 16 | Yorkshire | Boar | P | T | – | – | – | – | – | – | – | – | – | – | – | – | – | T | – | – | – | – | T | – | – | – | – | T | – | – | – | – | – | – | – | – | – |
| 17 | Tamworth | Sow | T | – | – | – | – | + | P | T | – | – | T | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| 18 | Duroc | Gilt | + | + | + | P | T | + | + | P | T | – | T | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| 19 | Berkshire | Gilt | + | + | + | P | T | + | + | + | + | P | + | + | + | + | + | P | P | T | – | – | – | – | – | – | – | | | | | | | | | | |
| 20 | Berkshire | Gilt | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | P | P | T | – | – | – | – | – | – | – | | | | | | | | | | |
| 21 | Duroc | Boar | | | | | | P | T | – | – | – | P | T | – | – | – | + | + | + | + | + | + | + | + | P | T | P | T | – | – | – | – | – | – | – | – |
| 22 | Chester | Boar | | | | | | + | + | P | T | – | + | P | P | T | – | + | P | T | – | – | P | P | T | – | – | – | – | – | – | – | – | – | – | – | – |
| 23 | Berkshire | Boar | P | P | T | – | – | + | + | P | T | – | + | + | P | T | – | + | + | P | T | – | + | + | P | T | – | T | – | – | – | – | – | – | – | – | – |

of this state, first by means of the agglutination test. The tabulated results of this survey are presented in table IV. The survey was made on a total of 1672 hogs in 15 herds. The number of hogs in each herd varied from 17 to 238. The herds were selected indiscriminately. We had had no previous information that Brucella infection was present in any of them.

TABLE IV—*Field survey of Brucella infection in swine in Michigan*

| HERD | HOGS TESTED | NEGATIVE | NUMBER REACTING IN A DILUTION OF: | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | 1:25 | | | 1:50 | 1:100 | 1:200 | 1:500 |
| | | | T | P | + | + | + | + | + |
| 1 | 33 | 20 | 4 | 3 | 5 | 0 | 0 | 0 | 1 |
| 2 | 17 | 13 | 0 | 0 | 4 | 0 | 0 | 0 | 0 |
| 3 | 238 | 181 | 6 | 20 | 30 | 1 | 0 | 0 | 0 |
| 4 | 53 | 40 | 1 | 5 | 4 | 1 | 0 | 1 | 1 |
| 5 | 193 | 99 | 15 | 17 | 19 | 10 | 9 | 2 | 22 |
| 6 | 25 | 20 | 0 | 0 | 1 | 0 | 0 | 1 | 1 |
| 7 | 28 | 21 | 1 | 2 | 3 | 1 | 0 | 0 | 0 |
| 8 | 99 | 88 | 1 | 2 | 3 | 0 | 1 | 1 | 3 |
| 9 | 62 | 38 | 3 | 9 | 11 | 1 | 0 | 0 | 0 |
| 10 | 199 | 164 | 8 | 14 | 12 | 0 | 1 | 0 | 0 |
| 11 | 132 | 97 | 2 | 15 | 1 | 1 | 2 | 0 | 0 |
| 12 | 77 | 66 | 0 | 2 | 9 | 0 | 0 | 0 | 0 |
| 13 | 132 | 53 | 19 | 12 | 7 | 15 | 8 | 5 | 13 |
| 14 | 163 | 153 | 2 | 1 | 5 | 0 | 0 | 0 | 0 |
| 15 | 221 | 147 | 14 | 15 | 15 | 8 | 9 | 5 | 8 |
| Total | 1672 | 1202 | 143 | 117 | 76 | 38 | 32 | 15 | 49 |
| Percentage | | 71.9 | 8.6 | 7.0 | 4.5 | 2.3 | 1.9 | .9 | 2.9 |

The serological data show that not a single herd was encountered in which there was not found at least a few animals reacting to the agglutination test in one of the titres employed. In a few herds (2, 12 and 14) only a comparatively small number of hogs reacted to the test, and these in low titres. In two herds (5 and 13) 17 and 31 per cent, respectively, of the total number tested reacted in titres of 1:50 and above. The percentages of the total number of animals reacting in each of the titres employed are not high figures. One would expect to find such low percentages if the serological data obtained in the first herd studied have any significance. That is, one would expect to find herds in which the agglutination titres of the animals are falling or disappearing, and herds containing animals with rising and high titres. In fact one of the herds in the survey, when tested several

months later, showed a pronounced reduction in the number of animals reacting in a high titre

From each herd included in the survey, we sought information as to the occurrence of abortions in the brood sows. We were informed by a few of the herd-owners that occasionally a sow aborted, but no serious losses had occurred. Certain herd-owners did mention, however, that considerable trouble had been experienced in getting brood sows and gilts to conceive.

## Discussion

We have concentrated our studies chiefly on one particular herd of hogs of sufficient size with the view of learning more about the natural course of Brucella infection, its nature and the location of the organism within the body of the hog. In the course of the study we have encountered 62 animals in the one particular herd reacting to the agglutination test in low and in high dilutions. Of those that showed agglutinins in their blood during the study, 38 were slaughtered and one died. Of the 128 animals that consistently failed to show evidence of agglutinins in their blood, 15 were slaughtered. A thorough search was made of all internal organs, lymph-nodes, muscle tissues, blood and in some cases portions of the contents of the intestines, for the presence of *Br. suis* in all animals, both reacting and non-reacting, that were slaughtered. Of the total number examined bacteriologically, the organism was recovered from only 15 animals. The habitats of the organism in these animals are summarized in table V. Though the spleen and lymph-nodes appear to be the chief location of *Br. suis*, it may occasionally be found in other parts of the body as well. Graham, Boughton and Tunnicliff[12] also report the finding of the organism in the visceral lymph-nodes of one naturally infected sow, while Connaway, Durant and

Table V—*Location of Brucella suis in tissues of 15 naturally infected hogs*

| Tissue | Hogs Giving Positive Culture |
|---|---|
| Gastric lymph-nodes | 4 |
| Spleen | 11 |
| Supramammary lymph-nodes | 4 |
| Internal iliac nodes | 2 |
| Submaxillary nodes | 2 |
| Kidneys | 2 |
| Uterus | 1 |
| Udder | 1 |
| Liver | 1 |

Newman[7] and Weeter[13] have isolated *Br. suis* from the non-gravid uterus and the milk of such animals.

All of the cultures of Brucella isolated from the 15 hogs set forth in table I and from three other hogs in one of the field herds grew out aerobically. When they were studied as to species,[12] all were found to be *Br. suis*. The pathogenicity of strains infecting the hogs could hardly be questioned, since two men who handled the hogs from time to time became infected with *Br. suis*, showing marked symptoms of undulant fever. The time of onset in one case was December, 1929, in the other, January, 1930

The disappearance of agglutinins altogether, or their tendency to disappear in 45 of 62 hogs that reacted to the agglutination test, would strongly suggest that the organism in most instances does not remain in the body of the hog longer than from 3 to 5 months. If this be true, the elimination of the infection in a herd of hogs becomes a very simple procedure and may be accomplished without making unnecessary sacrifices of a large number of animals. The elimination of the disease from a herd may be accomplished by separating all reacting animals from the non-reacting, immediately after the first blood test. Monthly tests should be continued on those that were negative in the first test for at least three months, in order to detect new reactors. At the end of five months, the original reactors should be tested again. Those in which agglutinins still persist should be eliminated from the herd. The procedure first described has been followed in the elimination of the disease from the large herd discussed in this paper. No new reactors have appeared in the herd for several months and, with few exceptions, all the reacting animals that were not slaughtered are now negative.

In a search of the literature we find that Hayes and Phipps[14] followed the course of the disease in a naturally infected herd of 60 hogs by means of the agglutination test. In the herd which they studied it appears that there was a tendency for more animals to react over a longer period of time than in the one we have studied. However, they noted that there was a general tendency for the agglutination titres of the animals to fall after one year. This was true also in the case of the 17 naturally infected hogs studied by Graham, Boughton and Tunnicliff.[10]

The data relative to the breeding and farrowing history of the herd under study furnish some very interesting information on the influence of Brucella infection on the breeding efficiency of

brood sows and gilts. Out of a total of 48 sows and gilts bred in the fall of 1929, 10 failed to farrow, due to sterility or early unobserved abortion, and one aborted 6 fetuses. *Br. suis* was recovered from the organs of 9 of the 10 animals failing to breed and the one that aborted at the time they were slaughtered. The remaining 37 sows and gilts delivered at farrowing time 350 living and 44 dead pigs. Of the living pigs, 153 died from miscellaneous causes before they were weaned.

A thorough bacteriological examination was made of the internal organs of a total of 109 pigs expelled dead at the time of farrowing or died shortly after farrowing, in order to determine the possible relationship of *Br. suis* to their failure to survive. Of 100 dead pigs delivered by reacting sows and gilts, 35 showed streptococci of either alpha, beta or gamma type; 15, *Pseudomonas aeruginosa;* 3, *Escherichia coli;* and from the 6 that were aborted *Br. suis* was recovered in pure culture. From 9 dead pigs from non-reacting sows and gilts, 6 showed *Streptococcus gamma* and 4, *P. aeruginosa.* These data would indicate very strongly that there is little or no relationship between *Br. suis* infection, as evidenced by the presence of agglutinins in the blood of the sow, and the delivery of pigs, part of which are dead and part alive at farrowing time. It is evident that intercurrent infections, especially those due to streptococci and *P. aeruginosa,* play an important role in the causation of such an enormous loss of pigs at farrowing time. It is interesting to note that our findings coincide closely with those of Graham, Boughton and Tunnicliff,[10] who, on examining dead pigs from normally farrowing sows in a naturally infected herd, found a surprisingly large number of them infected with different organisms.

During the spring farrowing season of 1930, we procured, for examination for the presence of *Br. suis,* fetal membranes from 30 of the 38 sows and gilts that delivered living and dead pigs. This was accomplished through guinea-pig inoculations. The only fetal membranes from which *Br. suis* was recovered were those obtained from the aborting sow 13 (table I). These findings also parallel those of Graham and co-workers,[12] who failed to find *Br. suis* in 20 fetal membranes from sows in a naturally infected herd.

An effort was made to recover the organism from the blood and feces of reacting hogs through guinea-pig inoculations. Thus far, 100 blood specimens and 14 fecal samples have been examined. All were negative.

When reacting animals were slaughtered, we were careful to note such gross anatomical changes in the organs, which, if occurring constantly enough, might serve as a guide in identifying infected animals. The gross changes which were observed in the order of their constant occurrence were: spleen enlarged dorso-ventrally and ventral tip darkened, hemmorrhagic areas well distributed over its gastric surface; skeletal and visceral lymph-nodes congested; infarcts and areas of parenchymatous degeneration in kidneys; suppurative metritis with the presence of muco-purulent exudate; mild enteritis. There is a possibility that the gross changes which are seen in the spleen of the infected hog may serve as a means of identifying them from those that are not infected

The microscopic pathology of the infected tissues collected from the hogs at the time of slaughter is being studied by Dr. L. B. Sholl, of the Department of Pathology, and will be reported on later.

TABLE VI—*Comparison of results on hogs reacting to agglutination test conducted in three different states*

| REPORTED BY | STATE | HOGS TESTED | NUMBER OF REACTIONS IN DILUTION OF: | | | |
|---|---|---|---|---|---|---|
| | | | 1:20–25 | % | 1:40–60 | % |
| Hardy and associates[15] | Iowa | 343 | 98 | 28 | 74 | 21 |
| Boak and Carpenter[16] | New York | 4,014 | | | 64 | 1.54 |
| Johnson and Huddleson | Michigan | 1,672 | 336 | 19.9 | 134 | 7.99 |

In reviewing the field survey of Brucella infection in hogs in the state of Michigan it is interesting to compare the figures which we obtained with those of Hardy, Hudson and Jordan,[15] obtained on hogs slaughtered at a packing-house in Iowa, and those of Boak and Carpenter,[16] obtained in a similar manner, but in the state of New York. These comparisons are illustrated in table VI. It may be noted that the percentage of hogs which Hardy and associates found reacting to the agglutination test is much higher than the percentage found by Boak and Carpenter or ourselves. These comparative figures are very significant when one compares the incidence of undulant fever in three states. It is known to be higher in Iowa than in either New York or Michigan.

The field survey of Brucella infection in hogs indicates that it is widely disseminated in Michigan and more prevalent than our

past knowledge has indicated. In the past, we have always associated the presence of Brucella infection in hogs with the occurrence of abortions, and, since there have been but few reports of brood sows or gilts aborting during previous years, it was natural to assume that Brucella infection was not very prevalent in hogs in this state. Since breeders, with whom we have discussed the situation and in whose herds we have found many hogs reacting to the agglutination test and isolated *Br. suis* from them as well, have not observed their brood sows or gilts to abort, we are led to conclude that the disease in the hog cannot be considered as an abortion disease, such as we observe in cattle due to *Br. abortus* (Bang). From the gross changes found in the organs of infected hogs and the habitat of the organism in the tissues, the disease appears to be of the same nature as that found in the goat, due to *Br. melitensis* (Bruce). That is, it is a disease chiefly of the lymphatic tissues.

In the course of the examination of fetal membranes from 30 sows and gilts, we noticed that on many of them there were irregular glistening white deposits varying from 5 to 150 millimeters in diameter. These deposits appear like white enamel paint. Connaway, Durant and Newman[7] appear to have been the first to describe the occurrence of these deposits in the fetal membranes from hogs. They were unable to associate *Br. suis* with this particular formation. We also were unable to find *Br. suis* in the deposits. In the fetal membranes from sow 13 (table I) that aborted and from which *Br. suis* was recovered, were seen smooth, leathery, yellowish-brown areas. These areas were identical in appearance with those often seen in the interplacental spaces in fetal membranes of cattle infected with *Br. abortus* Bang).

In considering the bearing which the foregoing data may have on the problem of human infection, two thoughts suggest themselves. One is, that the wide prevalence of the disease in hogs and the manner in which they are handled on farms and in markets furnish a fertile source for infection in humans; the other is, from the course and nature of the disease in hogs, no considerable difficulty should be encountered in suppressing it and eliminating it as a source for infection in humans.

The writers wish to express their thanks and indebtedness to Mr. V. A. Freeman and Mr. G. E. Garn, of the Animal Husbandry Department of the College, for the coöperation and assistance which they have given us during the course of this study.

## Summary

The findings set forth in this paper may be summarized as follows:

Brucella infection in swine appears to be due to one species of Brucella, namely, *Br. suis* (Traum).

The rapid agglutination test is an accurate method of following the course of Brucella infection in swine.

Brucella infection in swine appears to be a self-limiting disease, the majority of animals recovering at most within five months from the time agglutinins first appear in their blood.

Infected sows or gilts do not conceive as readily as those that are not infected.

The infection appears to be confined chiefly to the lymphatic tissues.

The capacity of *Br. suis* to invade the gravid uterus of sows or gilts does not appear to be marked.

The field survey indicates that the disease is very prevalent in hogs in the state of Michigan.


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## Rabies Quarantines

A 90-day quarantine was placed on all dogs in Iron County, Michigan, on April 1, following an outbreak of rabies. Berrien County, Michigan, was placed under a similar quarantine, April 13.


## B. HEMOLYTICUS INFECTION IN A HOG*

*By* EDWARD RECORDS AND MARTHA HUBER

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For about fifteen years bacillary hemoglobinuria (red water disease), an acute fatal disease due to infection with *B. hemolyticus*, has been under investigation at this Station.[1,2] At first, thought to be a disease of cattle only, sheep later were found to be highly susceptible when kept on infected pastures. Until very recently, however, we believed bacillary hemoglobinuria to be strictly a disease of ruminants under natural conditions. It would now appear that this conception is incorrect, in that at least one other species is susceptible to natural infection.

On January 3, 1931, Dr. F. E. Henderson, of Elko, Nevada, veterinary inspector for the State Board of Stock Commissioners, was asked to examine a hog which had been found dead by the owner that morning. The animal was apparently normal the night before. The owner suspected choking on some foreign body, as the animals were receiving some garbage.

On autopsy this hog presented a typical pathological picture of bacillary hemoglobinuria as it occurs in ruminants. There was deeply blood-colored fluid in the abdominal cavity. The usual severe generalized subserous hemorrhagic condition was present. Lymph-gland and kidney changes were characteristic, with port wine-colored urine in the bladder. The odor peculiar to this infection was well marked. Two of the peculiar infarcts, as far as known found only in this infection, were present in the liver, one at the periphery, the other near the center. In this point only did the lesions differ from those usually found in ruminants, where a single liver infarct is almost invariably found, though exceptions do occur.

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One of the liver infarcts, the spleen and several lymph-glands were packed in a liberal amount of borax and forwarded to this laboratory for examination. The specimens did not reach us until January 5, and work on them did not start until late that day, approximately 60 hours after the death of the animal.

The liver infarct only was examined bacteriologically. A section cut from the edge of the lesion, after searing the surface, was emulsified in a mortar with a small amount of normal saline solution. This emulsion was seeded into two series of freshly boiled and cooled tubes of peptic digest broth plus cooked ground beef heart, 0.2 per cent dextrose and 0.1 per cent dibasic potassium phosphate. Two series of freshly boiled and cooled peptic digest agar plus 0.2 per cent dextrose tubes also were seeded by the serial loop dilution method. All of the media used had a reaction of approximately pH 7.5. One series each of the broth and agar tubes were heated at 70° C. for six minutes and all four series incubated at 37° C.

A check smear made from the incised surface of the infarct section used for the emulsion showed a fair number of organisms typical of *B. hemolyticus* as it occurs in natural lesions.

A practically pure culture of *B. hemolyticus* developed in the heated and unheated series of both broth and agar tubes. Pure cultures were readily secured by picking colonies from the deep agar cultures after expelling same from the tubes and slicing them.

Two guinea pigs also were inoculated with 1 cc of the infarct emulsion. One died in about 60 hours and a pure culture of *B. hemolyticus* was recovered from the heart-blood. The second developed a local lesion, typical of infection with this organism, and recovered.

In addition to the cultural and pathogenic study, the identity of the organism recovered was confirmed by cross-agglutination tests. An agglutination antigen prepared from the recovered organism was completely agglutinated by ten known anti-*B. hemolyticus* sera in a dilution of 1:400 or higher. Using the same antigen, no agglutination was obtained with strongly agglutinating sera prepared from five other pathogenic anaerobes.

No satisfactory explanation of how this hog acquired the infection was arrived at. The group of hogs, of which this was one, was raised on a ranch about twenty miles from Elko, on a dry, well-drained side hill. Bacillary hemoglobinuria is not known to have occurred on this ranch, but cattle were lost from the disease on an adjoining ranch during the summer of 1930. The hogs were

moved to Elko, about ninety days before this case occurred, where they were kept in a dry feed-lot. The feed consisted of barley brought from the ranch where they were raised, plus a small amount of cooked garbage.

### Comment

To those interested in infection with *B. hemolyticus* and other pathogenic anaerobes this case presents several points of interest. The ready recovery of the organism from the liver infarct lesion sixty hours after death is in marked contrast to the usual findings in ruminants. In ruminants the recovery of *B. hemolyticus* much beyond twelve hours after death is difficult or impossible, early death of this organism in the tissues or overwhelming by saprophytes being the rule.

The establishment of the susceptibility of the hog to infection with *B. hemolyticus* may indicate that there is a wide range of susceptible hosts to this organism.

The rather close biologic similarity of the hog and man suggests the possibility that even the latter may be susceptible under certain conditions. Reasonable care to guard against possible infection by those working with bacillary hemoglobinuria and cultures of *B. hemolyticus* would at least appear in order.

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## CAPILLARIA ANNULATA IN HUNGARIAN PARTRIDGES*

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Three Hungarian partridges were presented for examination with the history that these birds were part of a flock of over a hundred which had been divided into pens of twenty-four. These birds appeared normal until about the first of March, 1931. At that time three birds from one pen showed indications of sickness, and died about April 14. All these birds were native raised.

About two days after the above birds were received, a Hungarian partridge from a different source was presented for examination. This bird was imported from Hungary, about February 22,

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1931, with four other partridges. This bird appeared to be in good health until about April 14, when it appeared sick and died on April 16.

Two of the first lot were very emaciated. One was found to have a cold in the head, with canker formation in the mouth. No parasites could be found in this bird. On opening the esophagus and crop of the other bird, a necrotic area was noticed at about the place where the esophagus dilates to form the crop. In this area there was a blood clot and the tissue surrounding it was thickened and the mucosa seemed to be partially destroyed. This section of the esophagus was drawn into a sort of pucker which almost closed the lumen. Parts of several very slender parasites were seen extending from this area and when the mucosa was scraped, large numbers of nematodes were brought to view. When the tissue was examined more in detail, these thread-like parasites were found in the mucosa, each one taking a tortuous course and had the appearance of having been sewed back and forth into the mucosa. There was a large number of these extending from the posterior portion of the esophagus to about the middle of the crop. All other organs in this bird appeared to be normal, so it was decided that these parasites were the sole source of the trouble in this bird.

The other partridge in this group was in good flesh and the cause of death was not determined. An examination of the esophagus and crop revealed two *Capillaria annulata* embedded in the mucosa of the crop. There was no macroscopic evidence of tissue change.

The partridge that had been imported from Hungary showed typical liver and cecal lesions of entero-hepatitis, and the cause of death was attributed to this. Several annulata were found in the mucosa of the crop but there was no visible macroscopic change in the tissue.

Cram[1] reports finding this parasite in turkeys, chickens and ruffed grouse and states that the parasite is apparently widespread in this country.

From the examination of these partridges it would appear that *Capillaria annulata* is a rather common parasite of this fowl, and when present in large numbers will cause enough damage to the tissue of the esophagus and crop to cause death in the infested fowl.

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## HYDROCEPHALUS IN A CALF

*By* L. B. SHOLL, *East Lansing, Mich.*

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The animal is a female Holstein calf, seven months old. She was apparently normal at birth. After removal from the dam she was put on an experimental ration. Blindness in the right eye developed. Disturbances of consciousness and equilibrium gradually developed and became quite marked. The temperature remained normal, and the appetite was good. The animal was killed for examination.

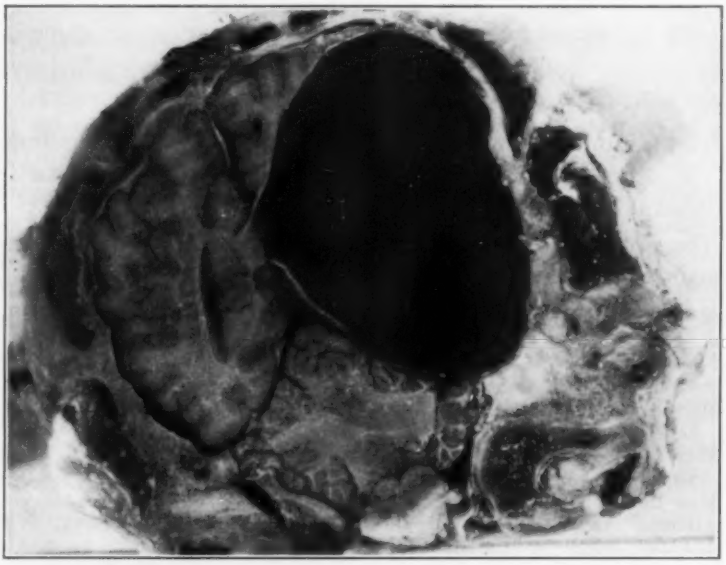

FIG. 1. Note the large cavity which has almost completely replaced the left hemisphere of the cerebrum.

The pupils of the eyes are dilated, especially that of the right eye. The cranial portion of the skull is somewhat deformed, the left side being somewhat larger than the right. The skull is cut so as to make a cross-section of the cranial cavity. There is a marked condition of unilateral hydrocephalus which has destroyed about ninety per cent of the left hemisphere of the cerebrum, has caused considerable bulging of the overlying bone, and is causing pressure on the cerebellum. The cavity is somewhat elliptical and is filled with a clear, watery fluid. This cavity

measures 8 cm. in length and 5 cm. in width. There is definite communication with the right lateral ventricle, but the right hemisphere of the brain appears normal except for some compression.

The findings in this case indicate the importance of examination of the head in cases showing symptoms referable to the brain, as many disease conditions may present nervous symptoms without definite gross lesions in the brain.

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## Empire State Veterinarians on the Air

Dr. H. K. Miller, of Mamaroneck, N. Y., president of the New York State Veterinary Medical Society, has appointed a Committee on Broadcasting to coöperate with Station WGY, of Schenectady, N. Y., in putting on a series of talks designed to give the veterinary profession some ethical publicity.

The Committee consists of Drs. J. G. Wills, Albany, chairman: F. W. Andrews, Mount Kisco; E. L. Brunett, Ithaca, Adolph Eichhorn, Pearl River, and J. J. Regan, New York.

The program was started the latter part of April and has been scheduled to run up to July 1, as follow:

April 20—"Your Local Veterinarian," Dr. J. G. Wills, Chief Veterinarian, N. Y. State Department of Agriculture and Markets, Albany.

April 27—"Care of the Pregnant Cow," Dr. J. N. Frost, N. Y. State Veterinary College, Ithaca.

May 4—"The Importance of Good Milk," Dr. J. J. Regan, Dairymen's League Co-operative Association, Inc., Middletown.

May 11—"Coccidiosis of the Chicken," Dr. E. L. Brunett, N. Y. State Veterinary College, Ithaca.

May 18—"The Value of Insulated Cooling Vats and How to Construct Them," Dr. F. D. Holford, Borden's Farm Products Company, New York.

May 25—"Cooling Milk on the Farm," Dr. C. I. Corbin, Sheffield Farms Company, New York.

June 1—"Fowl Pox," Dr. Norman J. Pyle, Pearl River.

June 8—"When the Cows Come Home," Dr. John McCartney, Borden's Farm Products Company, Middletown.

June 15—"How to Make Use of Your Veterinarian," Dr. H. K. Miller, president of the N. Y. State Veterinary Medical Society, Mamaroneck.

June 22—"Feeding Your Farm Animals," Dr. F. W. Andrews, former president of the N. Y. State Veterinary Medical Society, Mount Kisco.

July 1—"Odors and Flavors in Milk—Causes and Control," Dr. C. L. Kern, Dairymen's League Co-operative Association, Inc., Middletown.

Note that beginning with the week of June 29, the program will be given on Wednesdays, instead of Mondays. The broadcasts begin at noon.


RESPONSE OF INTACT SMALL INTESTINE IN NON-ANESTHETIZED DOGS TO CATHARTIC AGENTS, TO MORPHIN AND ATROPIN. Charles M. Gruber, William T. K. Bryan and Lyman K. Richardson. Proc. Soc. Exp. Biol. and Med., xxviii (1931), 5, p. 470.

The usual reaction of the intestinal loop to the cathartic drug when injected intravenously was a sudden decrease in the general tonus and a decrease in the height of the rhythmical contractions. Peristaltic contractions, if present, were temporarily inhibited, but contractions began two to ten minutes after injection of the drug. The amplitude increased gradually after becoming very severe. When the cathartic was placed in the lumen of the gut the first response was a rise of general tonus. This was followed by a decrease in general tonus to a point below the control level and by introduction of peristaltic contractions. When atropin was injected at the height of the cathartic action, there was always produced a temporary decrease in general tone and a disappearance of peristaltic contractions. Morphin sulfate, when injected intravenously, when injected into the lumen of the gut, or when given by mouth, invariably caused a sudden increase in general tonus of the gut and a temporary disappearance of peristaltic contractions.

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PARADYSENTERY IN YOUNG CHICKS. C. A. McGaughey. Jour. Comp. Path. & Therap., xliii (1930), 4, p. 308.

The author describes an outbreak of a disease in young chicks resembling pullorum disease. The disease did not spread to chicks hatched from other eggs. The organism isolated from the affected chicks appeared to be one of the group of paradysentery bacteria. It seems probable that the infection was transmitted through the egg as the disease was confined to one lot of chicks hatched from eggs which were bought from another breeder and also because the organism was recovered in pure culture from the

unabsorbed yolk in every case. The organism appeared to have a lower invasive power than *B. pullorum*.

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HEALING OF NECROTIC LESIONS PRODUCED BY BCG VACCINE. J. Zeyland. Abst. Arch. Path., xi (1931), 3, p. 486.

Necrotic lesions produced by BCG vaccine in heavy doses in animals have been noted by several authors. With such heavy doses killed organisms will of course produce identical tubercles with necrosis. A series of 23 rabbits was observed following the injection of BCG directly into the kidney for periods to 26 months. There appeared first necrosis around the wound, enclosing great masses of BCG surrounded by polymorphonuclears, some phagocytic. In two weeks there was a great necrotic area surrounded by typical tuberculous tissue, well vascularized. In the second month calcified areas appeared in the necrotic mass and the surrounding tissue lost its specific appearance. After a year various stages of healing occurred. The organisms were rare, the lesions small. Finally, healing appeared, virtually complete, with minor scar tissue. The benignity of the vaccine, even under the extreme conditions, seems assured.

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EFFECT OF BCG VACCINATION ON NEW BORN GUINEA PIGS BY MOUTH. A. I. Togunowa and M. M. Larionowa. Abst. Arch. Path., xi (1931), 3, p. 488.

Within the first few days after birth, guinea pigs received from 3.75 to 15 mg. of Calmette-Guérin bacilli by mouth. They developed normally. After from one to two months, about half of the animals had a temporary weak tuberculin allergy. Calmette-Guérin bacilli were demonstrable in the cervical and mesenteric lymph-nodes and they were resorbed within the first few days after the administration. None of the animals showed lesions of progressive tuberculosis. The vaccinated animals had no definitely demonstrable immunity to a definitely virulent reinfection.

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PROMOTION OF PHAGOCYTOSIS BY CALCIUM GLUCONATE, SODIUM IODID, DEXTROSE, AND OTHER SUBSTANCES. Ruth Tunnicliff. Jour. Inf. Dis., xlviii (1931), 2, p. 161.

Intravenous injection of solutions of calcium gluconate, calcium chlorid, sodium salicylate, sodium iodid, dextrose, and neosalvarsan into rabbits showed a marked increase in phagocytosis

by leukocytes in the blood. Physiologic solution of sodium chlorid, distilled water, milk, sodium phosphate, and mercurochrome in water or in dextrose, injected intravenously into rabbits, appeared to cause no increase in phagocytosis. Intravenous and intramuscular injections of calcium gluconate produced the same degree of phagocytosis in rabbits, but the former acted 24 hours earlier.

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Cutaneous Hypersensitiveness in Guinea Pigs Infected with Brucella Abortus. Axel C. S. Stroem. Jour. Inf. Dis., xlviii (1931), 2, p. 167.

Guinea pigs infected with Brucella organisms showed cutaneous hypersensitiveness to abortin (*Br. abortus* protein) even when gross anatomic lesions were absent. Guinea pigs infected with one strain of Brucella showed cutaneous hypersensitiveness to abortin prepared from five other strains. Guinea pigs inoculated with heat-killed vaccine or with a mixture of vaccine and kieselguhr did not show cutaneous hypersensitiveness to abortin. Guinea pigs infected with *Br. abortus* always gave negative reactions with tuberculin. Tuberculous guinea pigs gave slight, somewhat atypical reactions with abortin, presumably due to increased non-specific hypersensitiveness.

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The Blood Picture in Anaphylaxis. N. M. Nikolaeff and L. L. Goldberg. Physiol. Abst., xv (1931), 10, p. 619.

The peripheral blood of guinea pigs was investigated after the sensitizing injection, during the non-lethal shock later induced, and during the after period. In the differential count the leukocytes were divided into two classes: true leukocytes and histiocytes. During the period of sensitization and shock there occurs a large increase in histiocytes, and after the shock a marked fall accompanied by a large rise in eosinophiles. To some extent these cells appear to vary inversely in number. The general changes throughout the whole period of sensitization, shock, and recovery resemble those found during an acute infective process.

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Blood Studies in Hemorrhagic Anemia. H. S. Mayerson and Henry Laurens. Jour. Nutri., iii (1931), 5, p. 453.

The authors studied the blood cytology in severe secondary anemia produced and maintained for varying lengths of time in twenty-seven dogs following the method of Whipple and Rob-

scheit-Robbins. In dogs fed the standard bread S. there is an average weekly production of 4 gms. over and above the maintenance level. When apricots are added this becomes 15 gms., when lettuce is added, 10 gms., and with peaches, 7 gms. After several weeks of bread S. feeding there is a gradual increase in red cell number, accompanied by a decrease in the corpuscular volume and corpuscular hemoglobin. The percentage of hemoglobin in these smaller cells, however, remains relatively unchanged. If lettuce, apricots or peaches are added to the diet, these changes do not occur and they are interpreted as being compensatory reactions to the oxygen deficiency occasioned by the low hemoglobin when the severe anemic level is maintained.

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THE EFFECTS OF RADIANT ENERGY ON HEMORRHAGIC ANEMIA. Henry Laurens and H. S. Mayerson. Jour. Nutri., iii (1931), 5, p. 465.

Anemia was produced in twenty-four dogs employing the procedure and diet of Whipple and Robscheit-Robbins. Carbon and mercury arc irradiation of these animals results in marked and persistent increases in the number of erythrocytes and reticulocytes, the response being more marked with massive exposures than with smaller doses repeated more often. An increase in hemoglobin regeneration was noted in only one experiment. In twelve cases the amount of hemoglobin formed during and immediately following the irradiation period is much less than in previous control periods; in the remaining eleven experiments there was no change in the amount formed. The absence, under such irradiation, of any marked stimulus to hemoglobin formation is evidenced by the fact that peaches, apricots or lettuce, added to the diet of animals which had shown no response to radiation, produce their typical influence. The acceleration of hemoglobin regeneration produced by adding these substances to the diets also was not demonstrably influenced by any dosage of radiant energy as emitted from either of the sources used.

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THE OCCURRENCE IN CHICKS OF A PARALYSIS OF NUTRITIVE ORIGIN. L. C. Norris, G. F. Heuser, H. S. Wilgers, Jr. and A. T. Rengrose. Poultry Science, x (1931), 2, p. 93.

The occurrence of a peculiar form of paralysis in young chicks which appears to be caused by a deficient diet is described by the authors. The chicks suddenly lose partial control of the legs and

walk or rest on their hock joints. Later, if the paralyzed chicks do not die, a complete or partial spontaneous recovery is made. In the latter event a permanent paralysis of one or both feet persists and the chicks walk upon the distal end of the tarso-metatarsus or complete loss of the legs occurs and the bird lies upon the breast and abdomen with legs sprawled outward from the body. In all stages of this paralysis the leg muscles are flabby and withered. The hock joint is normal. The toes of the feet almost invariably curl inward and together. This nutritional paralysis has occurred on rations where commercial casein, purified casein, menhaden fish meal, meat scrap and dried whale meat have been used as the sole source of animal protein. It usually develops between the third and the tenth weeks of age, the number of cases varying from twenty to fifty per cent. The authors conclude that the causative factor is a vitamin deficiency. The factor is present in a vitamin concentrate from milk, yeast, autoclaved yeast and alfalfa meal. Meat scrap, menhaden fish meal and dried whale meat may be deficient in this factor.

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Incidence of Salmonella Pullorum in Eggs from Reactor Hens. Helen J. Weaver and John C. Weldin. Poultry Science, x (1931), 3, p. 118.

A study was made of 775 eggs laid over a period of three months by twenty reactor hens, 5 White Wyandottes, 5 Leghorns and 10 cross-breds of Light Brahma and White Leghorn. The birds were trap-nested and an effort was made to determine if there was any correlation between the percentage of infected eggs laid during the various phases of the clutch. The authors conclude that there is no such relation. Six birds did not lay an egg in which the organism was found.

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The Relation Between Fowl-Pox (Epithelioma Contagiosa) and Avian Diphtheria. I. J. Kligler and M. Aschner. Brit. Jour. Exp. Path., xii (1931), 1, p. 35.

Fowl-pox or contagious epithelioma and avian diphtheria are two distinct disease entities which may occur simultaneously or independently. There is no cross-immunity between these two affections. Fowl-pox virus may attack the tongue or oral mucosa with the production of ulcerative membranes. The membrane in this case is adherent, causes bleeding on removal, is leathery in consistency and circumscribed. Microscopically it is character-

ized by extensive necrosis of the epithelial tissue, infiltration or the presence of typical inclusion bodies. The diphtheritic membranes are irregular whitish or yellowish patches cheesy in consistency, and only slightly adherent. They do not contain fowl-pox virus. Histologically, they appear as superimposed layers on the epithelium which is only slightly injured. There is infiltration but little if any necrosis of tissue and no inclusion bodies are found. The type of lesion which we find due to avitaminosis is distinctly different. Macroscopically they have the appearance of whitish pustules scattered over the whole of the mucous surface of the pharynx. Microscopically the lesion is shown to be confined to the glands.

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THE ACIDITY PRODUCED IN BRUCELLA CULTURES. S. H. McNutt and Paul Purwin. Jour. Inf. Dis., xlviii (1931), 3, p. 292.

A study was made of some of the characteristics of the Brucella group of microörganisms in carbohydrate medium. Broth or peptone solution in which Brucella were grown without sugar became strongly alkaline. When dextrose, levulose, galactose, xylose or arabinose was added, the medium still became alkaline even though determinations of sugar showed a loss. When sugars were added to a solution of nutrose or a mixture of nutrose solution and serum, and the medium was inoculated with Brucella, very evident traces of acid were often produced in dextrose, levulose, galactose and xylose. With arabinose greater quantities of acid were formed. When Brucella were limited to a sugar as the only source of carbon, experiments indicate that growth took place in both arabinose and xylose. It was impossible to classify the strains according to source when grown in carbohydrate media.

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PULMONARY TUBERCULOSIS OF BOVINE ORIGIN. Editorial. Amer. Jour. Pub. Health, xxi (1931), 3, p. 280.

The author points out that one seldom now sees discussions of bovine tuberculosis in relation to human health. Some advocate pasteurization of milk, probably more on account of undulant fever than bovine tuberculosis. The stand of Koch is reviewed but data of prominent English bacteriologists are given to show that the bovine type of the organism can be the cause of pulmonary tuberculosis. The alimentary tract is indicated as the portal of entry. The author favors pasteurization of all market milk in addition to the efforts which are being made to eradicate tuberculosis from dairy herds.

## PUBLICATIONS RECEIVED

The Register of Veterinary Surgeons. Royal College of Veterinary Surgeons, London, 1931. pp. 391.

University of Pennsylvania Bulletin, School of Veterinary Medicine, Announcement 1931-1932, 47th Annual Session. Philadelphia, Pa., xxxi (1931), 17, pp. 48.

A Contribution to the Bacteriology and Pathology of the Bovine Udder. L. B. Sholl and J. P. Torrey. (Tech. Bul. 110. Agr. Exp. Sta., Mich. State Coll., East Lansing, Mich., January, 1931.) Illustrated. pp. 31.

Anaplasmosis in Cattle. Geo. W. Stiles, Jr. (Cir. 154. U. S. Dept. Agr., Washington, D. C., February, 1931.) Illustrated. pp. 10.

Transmission of Anaplasmosis by Flies. C. E. Sanborn, George W. Stiles, Lewis H. Moe and Harry W. Orr. Reprint from *Okla. Agr. Exp. Sta. 1930 Rpt.*, (1931) pp. 15.

Michigan State College of Agriculture and Applied Science, Announcements for the Year 1931-1932. East Lansing, Mich., 1931. pp. 349.

The Pharmacology of Thallium and Its Use in Rodent Control. James C. Munch and James Silver. (Tech. Bul. 238. U. S. Dept. Agr., Washington, D. C., April, 1931.) pp. 28.

Acute Poisoning from Bee Sting, Recovery. F. G. Cawston. Reprint from *Jour. Trop. Med. & Hyg.*, (1930) p. 1.

Pathological Conditions Associated with Schistosomiasis in South Africa. F. G. Cawston. Reprint from *Jour. Trop. Med. & Hyg.*, (1931) pp. 3.

Developmental Stages of Some Nematodes of the Spiruroidea Parasitic in Poultry and Game Birds. Eloise B. Cram. (Tech. Bul. 227. U. S. Dept. Agr., Washington, D. C., February, 1931.) Illustrated. pp. 27.

Nigeria, for the Year 1929, Fifth Annual Report of the Veterinary Laboratory, Vom, Northern Provinces. Lagos, Nigeria, 1931. pp. 84.

Johannesburg, Report of Director of Abattoir and Live Stock Market Department, 1st July, 1929, to 30th June, 1930, City of. Johannesburg, Union of South Africa, 1931. pp. 24.

Testicular Transplants in Goats. R. M. C. Gunn. Reprint from *Australian Vet. Jour.*, vi (1930), 2, pp. 56-65.

Testicular Grafts on Rams. R. M. C. Gunn and H. R. Seddon. Reprint from *Australian Vet. Jour.*, vi (1930), 3, pp. 104-112.

Testicular Grafts on Rams. R. M. C. Gunn and H. R. Seddon. Reprint from *Australian Vet. Jour.*, vi (1930), 4, pp. 132-145.

The Standardization of Biological Products. A. Eichhorn. (Presented at the Eleventh International Veterinary Congress, London, England, 1930.) pp. 15.

Fowl Paralysis. A. M. Lee and Cecil Elder. (Cir. 31. Wyo. Ext. Service, Laramie, Wyo., December, 1930.) Illustrated. pp. 10.

Nebraska State Veterinary Medical Association, Addresses and Papers Presented at Thirty-Third Annual Meeting. Lincoln, Nebr., December 9-10, 1930. E. C. Jones, Secretary, Grand Island, Nebr.

Kansas Veterinary Medical Association, Report of the Proceedings of the Twenty-Seventh Annual Meeting of the. Topeka, Kans., January 21-22, 1931. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kans. pp. 77.

Coccidiosis of Chickens. Cecil Elder and A. M. Lee. (Cir. 32. Wyo. Ext. Service, Laramie, Wyo., January, 1931.) Illustrated. pp. 8.

The Sterility of Catgut in Relation to Hospital Infections with an Effective Test for the Sterility of Catgut. Frank L. Meleney and Mabel Chatfield. Reprint from *Surg., Gynec. & Obst.*, lii (1931), pp. 430-441.

Salmon Poisoning. B. T. Simms, C. R. Donham and J. N. Shaw. Reprint from *Amer. Jour. Hyg.*, xiii (1931), 2, pp. 363-391.


## ARMY VETERINARY SERVICE

### Regular Army

Captain Forest L. Holycross is relieved from duty at Fort Riley, Kans., effective on or about May 15, 1931, and directed to proceed to Columbus, Ohio, for duty at the Ohio State University, and additional duty at Fort Hayes, Ohio; General Reserve Depot, Columbus, Ohio, and with the Organized Reserves of the Fifth Corps Area.

Captain Earl F. Long is relieved from duty at the Ohio State University and from additional duty as attending veterinarian, Fort Hayes, Ohio; and General Reserve Depot, Columbus, Ohio; from detail with the Organized Reserves of the Fifth Corps Area, effective on or about June 10, 1931, and directed to proceed to Fort Bliss, Texas, for duty.

Captain William H. Dean is relieved from duty as instructor at the Cavalry School, effective on or about June 10, 1931, and assigned to Fort Filey, Kans., for duty.

Captain George L. Caldwell is relieved from duty at Fort Oglethorpe, Ga., effective on or about June 15, 1931, and directed to proceed to Fort Riley, Kans., for duty.

Colonel William G. Turner, Presidio of San Francisco, Calif., has been directed to report to the president of the Army retiring board at headquarters Ninth Corps Area for examination.

### Veterinary Reserve Corps

*New Acceptances*

Mosher, Lawrence Adam....Capt.....1258 So. Oxford Rd., Atlanta, Ga.

*Promotions*

Ray, John Duncan.....To Capt.....1103 E. 47th St., Kansas City, Mo.

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### Notice of Examination for Appointment in the Veterinary Corps, Regular Army

Examination for the purpose of qualifying candidates for appointment in the Veterinary Corps, Regular Army, to fill existing vacancies, will be held within the continental limits of the United States, June 22 to 27, 1931, inclusive.

Application blanks, Form No. 62, A. G. O., may be obtained from The Adjutant General or The Surgeon General, U. S. Army, Washington, D. C., or from the Commanding Officer or Surgeon of any military post or station and, when completed, should be forwarded direct to The Adjutant General, U. S. Army, Washington, D. C.

# COMMENCEMENT

## ONTARIO VETERINARY COLLEGE

The following candidates received the degree of Bachelor of Veterinary Science at the Convocation of the University of Toronto, held on April 29, 1931:

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| Alme, H. | Henry, R. H. | Lockhart, J. R. |
| Bodendistel, J. J. | Holm, J. S. | Owers, A. E. |
| Brown, V. R. | Horsland, J. E. | Schmidt, R. L. |
| Burke, T. J. | Humphreys, F. A. | Siplock, N. L. |
| Eckert, A. F. | Hutson, L. R. | Stoneman, W. J. |
| Ferguson, H. F. | Jones, F. O. | Thompson, K. H. |
| Gendreau, L. A. | Lee, D. H. | |

Honors were awarded to members of the graduating class as follows:

*General Proficiency*

First Prize—V. R. Brown, of Harley, Ont.
Second Prize—L. R. Hutson, of Bridgetown, Barbados.
Third Prize—N. L. Siplock, of Middlefield, Ohio.

*Andrew Smith Memorial Medal*

V. R. Brown, of Harley, Ont.

*Helen Duncan McGilvray Honorarium*

L. R. Hutson, of Bridgetown, Barbados.

*Bacteriology*

Special Prize—N. L. Siplock, of Middlefield, Ohio.

*Diligence*

Special Prize—L. A. Gendreau, of Paquetteville, Que.

## K. S. C. Prizes

The eighth annual Recognition Day program of the Kansas State College took place in the College Auditorium, Manhattan, April 30, 1931. At that time all winners of student prizes in the College received their prizes and awards. The names of the winners of the prizes offered the students of the Division of Veterinary Medicine are as follows:

*General Proficiency*

(Prizes offered by Dr. E. A. Schmoker, K.S.A.C.'17, of Evanston, Ill.)
First Prize ($10.00).....Don Harvey Spangler
Second Prize ($5.00).....Andrew Lafayette McBride

*Therapeutics*

(Prizes offered by Jensen-Salsbery Laboratories, Kansas City, Mo.)
First Prize $10.00).....Lloyd Edwin Boley
Second Prize ($5.00).....John Lester George

*Pathology*

(Prize of $7.50 offered by the Veterinary Faculty of the College)
Andrew Lafayette McBride

*Physiology*

(Prize of $7.50 offered by the Veterinary Faculty of the College)
Richard Duncan Turk


## TWIN CITY VETERINARY MEDICAL ASSOCIATION

The February meeting of the Twin City Veterinary Medical Association was addressed by Dr. H. C. H. Kernkamp, of the University of Minnesota, on the subject of poultry diseases. His talk was illustrated with lantern-slides and charts, prepared from material obtained in the diagnosis laboratory of the University. A statistical report covered the results of more than 50,000 examinations of poultry specimens that had been submitted for diagnosis during the past 12 years. This number represented approximately 85 different diseases and pathological conditions occurring in poultry. It showed very clearly the seasonal trend of certain conditions. For example, tuberculosis occurs throughout the year, while pullorum disease and coccidiosis, as well as some of the parasitic ailments, vary according to the season of the year. The subject was discussed by Dr. R. Fenstermacher and Dr. C. P. Fitch. A large number of preserved pathological specimens helped to illustrate the subject further.

At the March meeting of the Association Dr. O. B. Morgan, of Minneapolis, who specializes in the diseases of small animals, was the principal speaker and demonstrator. He presented a number of cases in dogs and cats for medical diagnosis and treatment and later demonstrated the use of nembutal as an anesthetic for small animals, followed by some surgical operations. One of these was the catheterization of a male cat. Hysterectomies also were performed on a cat and a dog.

Both of these meetings were attended by about fifty veterinarians.

H. C. H. Kernkamp, *Secretary.*

## TRI-COUNTY VETERINARY ASSOCIATION

A meeting of the Tri-County Veterinary Association was held at Spring Valley, Minn., April 3, 1931. Dr. R. Fenstermacher, of the Division of Veterinary Medicine, University of Minnesota,

discussed the subject of brucelliasis in a very well prepared paper. The meeting was well attended and the veterinarians derived a great deal of interesting and instructive information from Dr. Fenstermacher's paper.

P. H. RIEDE, *Secretary.*

## VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held on Wednesday evening, April 1, 1931, in the Academy of Medicine Building, 103rd Street and Fifth Avenue. Dr. O. E. McKim called the meeting to order. The minutes of the March meeting were read and approved, subject to the insertion of the resolution adopted relative to raising the educational requirements for the practice of veterinary medicine.

Dr. Adolph Eichhorn, of Pearl River, was the speaker of the evening, and his topic was "Infectious Abortion and Its Relation to Undulant Fever in Man." He traced the early history of this disease, and showed how it is now causing more economic losses than tuberculosis did. The infection is spread by ingestion of the organism, genital discharges and experimentally by culture dropped in the eye. Nearly 12 per cent of all cows eliminate the organism in their milk, and the drinking of infected raw milk by people may produce undulant fever. There have been 200 cases reported in New York State and over 600 in Iowa, and it is on the increase. Dr. Eichhorn urged the testing of all cattle in the herd, the elimination of the reactors, the quarantining of new animals for six months before being introduced into the herd, and the vaccination of young heifers.

Dr. Eichhorn demonstrated the agglutination test and many members made the test for themselves, which is quickly made and can be done in the field. A rising vote of thanks was extended to Dr. Eichhorn for the interesting talk.

President McKim read a letter from Dr. Wynne in which he expressed his regret at being unable to attend our meeting.

It was regularly moved by Dr. C. S. Chase and seconded by Dr. H. K. Miller than an assessment of one dollar per year over and above the dues be laid upon the members of the Association, the money so collected to be a contribution to the International Veterinary Congress. The motion was carried and the Secretary

instructed to inform the American Veterinary Medical Association of this motion.

Dr. Chase read an Act of Assembly altering the reading of the law requiring an affidavit of the owner of a spayed dog. The law as amended would merely require a certificate from the veterinarian. The Secretary was instructed to wire the Committee on Agriculture, urging passage of Bill No. 1499.

Dr. E. B. Ackerman reported a case of a kidney "dropper" in a horse. Dr. R. S. MacKellar reported a case of inflammation of the brain in a horse. Dr. H. K. Miller reported a case in a dog where the esophagus had been torn off from the pharynx during a fight.

No further business appearing, the meeting adjourned.

JOHN E. CRAWFORD, *Secretary.*

## MICHIGAN-OHIO VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Michigan-Ohio Veterinary Medical Association was held at the Court House, Adrian, Michigan, May 14, 1931.

Dr. A. F. Schalk, of Ohio State University, and Dr. H. J. Stafseth, of Michigan State College, discussed everyday problems in connection with poultry diseases and suggested means of coping with some of the more common diseases of baby chicks.

Dr. B. J. Killham, Extension Specialist, Michigan State College, discussed what had been accomplished by the committees from the Michigan State Veterinary Medical Association and the College, in an effort to bring about a clearer conception of the function of county agents and extension specialists, so that the activities of these groups would not interfere with the work of the practicing veterinarian. Dr. Killham also related some very interesting experiences in connection with his first year's work in starting a state-wide program for the control of Bang's disease.

Dr. H. Preston Hoskins, secretary of the A. V. M. A., briefly reviewed the work already done by the A. V. M. A. Committee on Agricultural Extension Service and reported what was being done by several of the state associations to help the cause. Dr. Hoskins stated that it was his opinion that the ultimate solution of this rather vexing problem rested in the hands of the state associations and that the national organization could expect to

accomplish very little unless it had the active and united support of the various state associations.

The attendance at this meeting was the largest in several years. Twenty-one Michigan veterinarians were in attendance and eleven from Ohio. Among the latter were Dr. George Pierce, state veterinarian of Ohio, and Dr. A. J. DeFosset, in charge of tuberculosis eradication in Ohio for the U. S. Bureau of Animal Industry.

Officers for the ensuing year were elected as follows: President, Dr. A. H. DeGroot, Dundee, Mich.; vice-president, Dr. C. H. Hoffmire, Adrian, Mich.; secretary-treasurer, Dr. E. C. W. Schubel, Blissfield, Mich.

E. C. W. SCHUBEL, *Secretary.*

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## CONESTOGA VETERINARY CLUB

The Conestoga Veterinary Club held its eighteenth Annual Shad Supper at the Stockyards Inn, Lancaster, Pa., May 21, 1931.

Dr. R. C. Gross, of Elizabethtown, presided and a supper of planked shad was served to sixty-two members and friends from the University of Pennsylvania and from all the veterinary associations in the eastern part of Pennsylvania.

Dr. C. J. Marshall, Professor of Veterinary Medicine, University of Pennsylvania, outlined "The Early History of the Tuberculin Test," in the principal address of the evening. Many medical reports were read dating back to 1891, when the test was introduced. Doctor Marshall vividly described the skepticism existing at that time and touched on a historic summary of tuberculin testing being completed and to be reported at a later date. Dr. T. E. Munce, Director, Pennsylvania Bureau of Animal Industry, a collaborator in this work, then spoke on "The Public Health and Economic Importance of Tuberculosis Eradication." Reminiscences of early tests by Doctors J. B. Reidy, Henry W. Turner, E. C. Yoder and D. E. Hickman followed and then Dr. Paul V. Clarkson proved the progress of tuberculosis eradication in Pennsylvania with his statistics.

Other speakers included Dr. R. S. Amadon and Dr. M. A. Emmerson, of the University of Pennsylvania; Dr. M. F. Barnes, Director, Pennsylvania State Laboratory, and Dr. H. B. Mitchell,

president of the Lancaster Medical Club, who cited the death of a local woman through her care of a cow having Bang's disease.

The meeting was adjourned to meet June 4, 1931, at the Old Valley Inn, one mile east of York, Pa., on the Lincoln Highway, when a chicken and waffle dinner will be held in honor of the ladies.

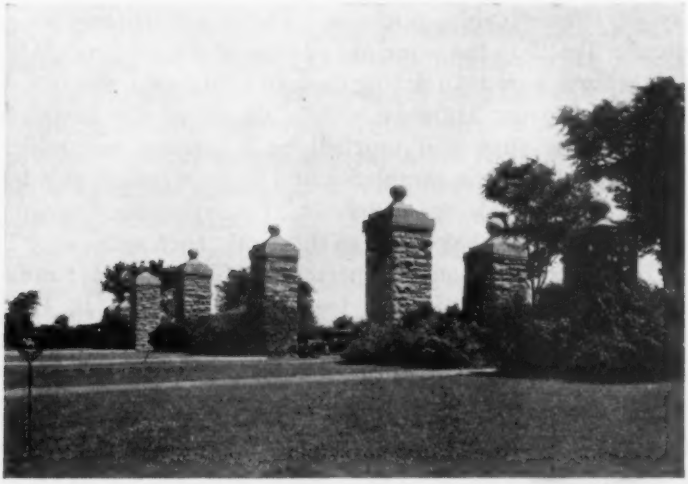
HENRY S. WEBER, *Secretary.*

## Missouri Veterinarians Confer with Extension Officials

A committee of five members of the Missouri State Veterinary Medical Association has been appointed to confer with the Extension Department of the University of Missouri in matters pertaining to coöperation between veterinary practitioners and extension workers throughout Missouri. This committee also will attempt to handle difficulties that arise implicating the veterinarian. The committee consists of Dr. Fred C. Cater, Sedalia, Chairman; Dr. J. L. Jones, Blackburn; Dr. W. S. O'Neal, Saint Charles; Dr. C. W. Strode, Ash Grove; and Dr. A. D. Glover, LaBelle.

## Name Changed

At the recent session of the Kansas Legislature, the name of the Kansas State Agricultural College was changed to Kansas State College of Agriculture and Applied Science.



Entrance to Swope Park, Kansas City,
third largest municipal park in the United States

# NECROLOGY

## FREDERICK CARL SCHMIDT

Dr. Fred C. Schmidt, of Portland, Oregon, died at his home, March 1, 1931, after an illness of several months.

Born in Leipzig, Germany, October 5, 1889, Dr. Schmidt received his preliminary education in his native land and his veterinary education in the United States and Canada. He first entered the St. Joseph Veterinary College, but finished his veterinary course at the Ontario Veterinary College in 1921.

Dr. Schmidt joined the A. V. M. A. in 1925. He was a member of the Oregon State Veterinary Association and the Willamette Valley Veterinary Medical Association. At the time of his death he was associated in practice with Dr. Wm. E. Ruggles (Colo. '27), in conducting the Portland Dog and Cat Hospital. Surviving Dr. Schmidt are his widow, two sons, two brothers and a sister.

W. E. R.

## GEORGE A. MEYERS

Dr. George A. Myers, of Albion, Pa., a registered non-graduate practitioner (Pa. 828) died April 22, 1931, of pneumonia. He was 68 years of age and had been a resident of Albion for the past 25 years. He was a member of the Conneaut Township Board of Supervisors, at the time of his death. He is survived by his widow and four children.

## H. E. HIGGINS

Dr. H. E. Higgins, of Shelby, Ohio, died at his home, April 23, 1931, after an illness of about six months. He was 58 years of age. He had practiced at Shelby continuously, ever since his graduation from the Ontario Veterinary College, in 1907. He is survived by his widow, seven daughters and four sons.

## WILLIAM HERBERT SIMMONS

Dr. William H. Simmons, of Louisville, Ky., died at his home, April 24, 1931. He was 66 years of age.

Born in England, Dr. Simmons received his veterinary education at the Ontario Veterinary College. Following his graduation, in 1892, he served in the British Army, in India, for a number of years. On December 16, 1920, he was appointed State Veterinarian of Kentucky and served until July 1, 1926, when he was succeeded by Dr. D. E. Westmorland. For the past five years Dr. Simmons had been veterinarian at the Bourbon Stockyards, Louisville.

Dr. Simmons joined the A.V.M.A. in 1912. He was actively indentified with the U. S. Live Stock Sanitary Association while State Veterinarian of Kentucky and served as a vice-president for two years (1921-22 and 1925–26). Surviving the deceased are his widow, one son, three stepchildren, four sisters and two brothers. Interment was at Lexington.

## FREDDIE LOUIS BEAR

Dr. Freddie L. Bear, of Charleston, Ill., died at his home, April 25, 1931, after a protracted illness.

Born at Effingham, Ill., September 6, 1878, Dr. Bear attended local schools and then entered the Terre Haute Veterinary College. Following his graduation in 1912, he located at Effingham, but removed to Charleston four years later. In August, 1917, he entered the Veterinary Corps of the U. S. Army as second lieutenant and saw two years of service. It is believed that the affliction, which finally resulted in his death, was contracted during his military service.

Dr. Bear joined the A. V. M. A. in 1912. He held membership in the Odd Fellows, the Elks, and the American Legion. He is survived by his widow, one daughter, two sisters and two brothers.

## JOACHIM F. SCHUBERT

Dr. Joachim F. Schubert, of Tampa, Florida, was murdered under mysterious circumstances the night of April 26, 1931. He received a call from outside the city and failed to return. Later the police found his body in a field near the outskirts of Tampa. He had apparently been slain with an ax and the body covered

with a tarpaulin. Dr. Schubert was born in Germany, attended grade schools in that country and then came to the United States. He was graduated from the New York State Veterinary College at New York University in 1917. One year later he entered the employ of the Lederle Laboratories at Pearl River, N. Y., and remained with that organization until 1924, when he went to Florida and located at Tampa, in general practice.

## DAVID W. MARKS

Dr. David W. Marks, of Chicago, Ill., died in St. Joseph's Hospital, April 28, 1931, after a long illness. He was 46 years old. Dr. Marks was a graduate of the Chicago Veterinary College, class of 1910, and was an officer of the Marks Expressing and Teaming Company, of Chicago.

## ORVILLE L. BOOR

Dr. Orville L. Boor, of Muncie, Indiana, died May 7, 1931, at the age of 71 years. Death was due to a heart attack and occurred suddenly.

Following his graduation from the Ontario Veterinary College, in 1890, Dr. Boor spent two years in the service of the U. S. Bureau of Animal Industry, on meat inspection. In 1892 he located at Muncie and engaged in general practice there until April 1, 1919, when he retired to spend the rest of his life at leisure. Dr. H. Meade Hamilton took over his practice and hospital.

Dr. Boor joined the A. V. M. A. in 1907. He was a member of the Publication Committee, 1908-09; a member of the Committee on Intelligence and Education, 1911-12, and chairman of the Necrology Committee, 1912-13. He was a member of the Indiana State Veterinary Medical Association and a tireless worker for veterinary legislation in the Hoosier State. For many years he served as secretary of the Indiana State Veterinary Medical Examining Board, and was professor of veterinary materia medica in the Indiana Veterinary College for several years. In civil affairs Dr. Boor always took a deep interest. He was active in the local Red Cross, the Tuberculosis Society and the Muncie Humane Society. He served all of these organizations as president and director at various times. He was a very active and loyal member of the Kiwanis Club, and his lodge affiliations included the Masons and Knights of Pythias.

Although Dr. Boor had been out of active practice for the past twelve years, his thoughts and interest continued to be with his chosen profession. He was honored and held in high esteem by everybody who knew him. He is survived by his widow. An only child, a son, died in 1911.

H. M. H.

## JOHN B. STEVENS

Dr. John B. Stevens, of Yale, Mich., died at his home, May 10, 1931, after an illness of only a few hours.

Born at Stouffville, Ontario, October 2, 1857, Dr. Stevens attended the Ontario Veterinary College and was graduated in 1888. Three years later he located at Yale and practiced there until the day before his death. He served as mayor of Yale for two terms and as councilman for several years.

Dr. Stevens was one of a family of veterinarians. One brother Dr. James B. Stevens (Ont. '88), of Toronto, is still living, while another brother, Dr. William Stevens (Ont. '85), formerly of St. Marys, Ont., preceded him in death. His elder son, Dr. C. C. Stevens (Ont. '04), is city veterinarian and dairy inspector of Port Huron, Mich., while the younger son, Dr. Walker W. Stevens (Ont. '11), of Toronto, is employed in the Department of Agriculture. His son-in-law, Dr. Morgan J. Smead (Ont. '10) is located at Rochester, Mich., at the Parksdale Farm of Parke, Davis and Company.

# PERSONALS

## BIRTH

To Dr. and Mrs. Jesse A. Jones, of Los Angeles, Calif., a daughter, March 28, 1931.

## PERSONALS

Dr. G. E. Bowler (Mich. '30) is located at Tecumseh, Mich.

Dr. Walter Peterson (Iowa '30) has located at Osceola, Wis., for general practice.

Dr. W. E. Russell (Chi. '20) has removed from Northam, P. E. I., to Kitchener, Ont.

Dr. C. L. Campbell (O. S. U. '26) has removed from Decatur, Ill., to South Bend, Ind.

Dr. W. F. Fisher (Colo. '23), formerly of Fallon, Nev., is now located at Elko, Nev.

Dr. Cliff D. Carpenter (Corn. '20) is a director of the Rotary Club of Van Nuys, Calif.

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Dr. L. B. Wolcott (K. S. C. '12) has removed from Grand Island, Nebr., to Shelton, Nebr.

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Dr. W. C. Hanawalt (Chi. '92), formerly of Payson, Ill., is now located at Hull, Ill., in general practice.

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Dr. L. V. Puckett (Ind. '18), formerly of Quincy, Ill., is now located at 215 S. 11th St., Mount Vernon, Ill.

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Dr. W. H. Busic (O. S. U. '29), formerly of Susanville, Calif., is now located at 1108 Park Ave., Chico, Calif.

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Dr. Homer A. Dennewitz (Geo. Wash. '18) has located at Leesburg, Ohio, where he is now engaged in general practice.

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Dr. Cass J. Kershaw (Mich. '30) has accepted a position as assistant to Dr. E. R. Steel (Corn. '14), of Kansas City, Mo.

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Dr. H. E. Erickson (Ont. '07), of Milledgeville, Ill., was elected president of that village at the election held the latter part of April.

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Dr. L. B. Wolcott (K. S. A. C. '12) has removed from Grand Island, Nebr., to Shelton, Nebr., where he is now engaged in general practice.

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Dr. John F. Deadman (Det. '95), of Sault Ste. Marie, Mich., has returned to his home, after a trip that took him as far south as Demopolis, Ala.

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Dr. Erwin Jungherr (Vienna '22) has accepted the position of veterinary pathologist at the Storrs (Conn.) Agricultural Experiment Station.

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Dr. Mark Welsh (Mich. '19) has been acting inspector in charge of hog cholera work, in Maryland, since the death of Dr. I. K. Atherton, in January.

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Dr. Michael D. Ducey (Ont. '16), formerly of Merrill, Mich., is now in Detroit, Mich., at 23 Cedarhurst Ave. He is with the Detroit Board of Health.

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Dr. C. C. Wagner (O. S. U. '26), of Cleveland, Ohio, recently returned to his home, after a stay in Asheville, N. C., where he was a patient at one of the hospitals.

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Dr. John P. Hutton (O. S. U. '11), of East Lansing, Mich., was secretary of the ninth annual Michigan State College R. O. T. C. Horse Show, held May 29-30, 1931.

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Dr. Andrew C. Merrick (O. S. U. '24), of West Jefferson, Ohio, has removed to Cleveland, where he has accepted a position as manager of a veterinary hospital in that city.

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Dr. T. M. Dick (O. S. U. '16) was recently reappointed city veterinarian of Little Rock, Ark. Dr. Dick has held this position for about ten years, to the satisfaction of all concerned.

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Dr. M. R. Sebright (St. Jos. '16), of Hartington, Nebr., has received an appointment in the U. S. Bureau of Animal Industry, and has been assigned to meat inspection at South Saint Paul.

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Dr. L. R. Twete (O. S. U. '22), of Thief River Falls, Minn., has opened a drug store at Saint Hilaire, Minn., operated by an assistant pharmacist. Dr. Twete is a graduate pharmacist as well as veterinarian.

Dr. W. A. Anderson (K. C. V. C. '07), of Sleepy Eye, Minn., has been appointed a member of the Minnesota State Live Stock Sanitary Board, to fill the vacancy created by the death of Dr. H. A. Greaves, recently.

Dr. H. V. Lewis (Iowa '29) has resigned his position at the South Dakota State College to enter into general practice with Dr. F. H. Hasenmiller (Chi. '03), at 1619 W. Locust St., Davenport, Iowa, on or about June 10.

Dr. Wm. J. Lentz (U. P. '04), Director of the Small-Animal Clinic at the University of Pennsylvania Veterinary School, addressed the meeting of the College of Physicians, at Philadelphia, April 29, 1931, on the subject, "Rabies in the Dog."

Dr. T. P. White (K. C. V. C. '11), of the Division of Hog Cholera Control, U. S. Bureau of Animal Industry, has been detailed from the Department of Agriculture as a member of the Board of Examiners of the U. S. Civil Service Commission.

Dr. John D. Beck (U. P. '28), of the veterinary faculty at the University of Pennsylvania, left for Europe on April 3, where he will make a special study of veterinary diagnosis at a number of the European veterinary schools. He will return September 1.

Dr. J. A. Allen (Ont. '16), for some time employed by the fox-breeders of Manitoba, has been appointed superintendent of research work and is attached to the Game and Fisheries Branch, Department of Mines and Natural Resources, province of Manitoba, and is located in Winnipeg.

Dr. L. E. Starr (O. S. U. '13) has been granted a leave of absence from the Virginia Polytechnic Institute, from September 1, 1931, to June 1, 1932, to work for his Ph.D. degree in the School of Medicine at the University of Virginia. He will major in pathology and bacteriology. Dr. Starr received his M. S. from Ohio State University in 1922.

THE PIONEER MOTHER
A statue group in Penn Valley Park, near Union Station, Kansas City.